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PHYTOPATHOLOGY

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ETIOLOGICAL PHYTOPATHOLOGY

GEORGE K. K. LINK

(Accepted for publication Dec. 2, 1932)

I. CONCEPTS, SCOPE, AND RÔLE

Like other experimental sciences, phytopathology aims to establish by experiment, that is, by deliberate change of one factor at a time, the simultaneous and serial antecedents out of which a pathic event (12) arises at a given time and place within an affected plant. It aims to analyze into their constituent part events complex total pathic events, such as an infectious disease, that occur during or follow development, and to establish the factors out of which each part event in the series arises. In so far as these factors are antecedent to, *i.e.*, condition and/or incite, pathic events, they are pathogenic factors (12). In this process of analysis or detailed description, stimulus-reaction chains or chains of causation are established in which the initial stimulus and the final reaction often are separated temporally and spatially by a long series of intermediate reactions and stimuli. When all of the series of causation chains of an event and their interrelations have been completely established, each part event, and, consequently, the complex total event, is said to be causally explained. In practice a causal explanation merely means that attention has been focused upon one part of the complex, which is called the effect, and then upon a striking feature of the complex of factors antecedent to it, which is called the cause.[1]

The branch of plant pathology that concerns itself with the causal analysis of pathic events has long been designated as etiology or etiological pathology. The term etiology[2] is derived from aitia (*αἰτία*), which means

[1] According to the present conception of the principle of causality, experiment is not able to establish absolute, invariant relations between so-called antecedents and consequents. Absolute predictability of events consequently is no longer considered the goal or even the ideal of science. Instead, science aims to establish by experiment the probabilities of apparent relations of events. While this conception is not a new one in the history of science, the advent of the new experimental and theoretical physics has been largely instrumental in bringing it into prominence.

[2] Democritus (5th century B. C.) was the first to use the term etiology. While it and its adjective, etiological, do not appear in the only treatise on the healthy and pathic behavior of plants that has come down to us from Greek antiquity, the term *aitia* is used throughout this discourse and in its title. This inquiry (27) by Theophrastus

blame or cause, and from logia (λογία), which means a reasoned discourse, an inquiry. Consequently, etiology is a system of knowledge or an inquiry dealing with the antecedents of events and with the rational study of the concepts in which the relations of antecedent and consequent are expressed.

By a shift, the term etiology has come also to mean the totality of the antecedents of an event, rather than the body of knowledge pertaining to these antecedents. The adjective etiological has experienced a similar shift in meaning. Thus the phrase "the etiology of mosaic" when used in this sense means the complex of antecedents out of which mosaic arises.

By a further shift the term etiology has been restricted to the obvious external antecedent of a pathic event, or to the body of knowledge dealing with this obvious causal factor. This factor is often designated as *the cause*, with an implication that it is the *sole cause* (12). In many text-books and special treatises the category of etiology includes only the specific pathogenic microorganism when infectious diseases are discussed, or the obvious external factor when noninfectious diseases are the object of inquiry. The specific pathogenic microorganism is often designated as *the pathogen*.

(370–286 B. C.) bears the title (ABOUT THE) CAUSES OF PLANTS (*Peri Phyton Aition*). Etiological pathology therefore can trace its lineage to the "Father of Botany."

By the end of the 2nd century A. D. the terms etiology and etiological had begun to have technical meanings. Galen (130–200 A. D.) in his classification of the branches of medicine (11) stated: "The etiological, which is also called pathological, is that part (of medicine) in which we examine that which happens against nature and investigate the causes of disease and the concourses of symptoms and inquire closely into the conditions of affections." Incidentally, this is Galen's definition of the field of pathology. While Galen defined the field of the pathological, he did not introduce the term pathology. Pathology had not yet been recognized as a distinct science. The word is not found in Greek lexica. Its Latin form, *pathologia*, had made its appearance by the 6th century A. D. and occurs for the first time in Corpus Glossariorum Latinorum (ed. Götz) IV, 263, 3; V, 384, 47, and is defined as *ratio passionis*, a reasoned discourse on affections. According to Galen, pathology is that part of medicine that concerns itself with the causal analysis of the subject matter of medicine. This still is the current definition of the field of human pathology. Phytopathology, historically and currently, is the botanical equivalent of medicine and hygiene and includes not only etiology but diagnosis (symptomatology) and preventive and remedial control (plant protection) as well.

Other biological sciences, such as physiology and genetics, although engaged in description of the probable sequences of relations of their subject matter, no longer recognize the etiological approach by a distinctive term. As theoretical and experimental phytopathology becomes a mature natural science, it too will become so thoroughly etiological that it probably will cease to designate its etiological descriptions by a differentiating term. Hence, the terms etiology and etiological probably will disappear from the vocabulary of theoretical pathology just as they have been dropped from the vocabularies of physics, chemistry, and physiology.

This highly limited and artificial conception of etiology and the fact that any scientific analysis that is more than a description of the static aspects of events concerns itself with conceptual and experimental analysis of the antecedents of events have been important factors in focusing the attention of phytopathologists upon pathogenic bacteria and fungi in the causal analysis of plant diseases. As a consequence, phytopathological activity and literature have been so largely concerned with the taxonomy, morphology, cytology, physiology, ecology, and genetics of pathogenic microorganisms, especially fungi, that phytopathology now is regarded as essentially parasitological mycology.

Recently, Morstatt (14) and Whetzel (26) have protested against the dominance of etiology in phytopathology. Morstatt contends that the etiological point of view stands in the way of a thoroughgoing general phytopathology because it has forced the pathic plant with its disturbed structures and processes out of the center of interest and consideration and thereby arrested the development of cellular phytopathology, which he considers the basis for a general phytopathology. He asserts further that the dominance of etiology has split the field of phytopathology into an applied botany which is restricted to applied mycology, and into an applied zoology, which is limited to applied entomology (13), and thereby retarded development of a general phytopathology. Finally, he contends that this preoccupation with eitology has led to neglect of those ecological factors, other than the parasite, of the pathic plant that play a rôle in parasitic diseases and to an almost complete disregard of nonparasitic diseases that constitute the best approach to a general phytopathology.

Whetzel (26) protests against the dominance of the etiological point of view because he contends that it has been responsible for the preoccupation of phytopathologists with fungi and bacteria and for their neglect of anatomical phytopathology. His stricture on etiology is not surprising in view of his definition of etiology as "that phase in the study or discussion of a disease which deals with the chief causal factor, its nature, classification, and relations with the suscept." Suscept, in turn, is defined as "any plant liable to infection by a given pathogene," and pathogene as "any organism capable of causing disease" (25).

Reflection upon these strictures against etiology and its deleterious influences reveals that they do not apply to a *thoroughgoing etiology*, but only to the *pseudo-etiology*, which has dominated phytopathology since the last half of the 19th century. This false etiology arose as a consequence of the quest for single external causes, which dominated the atomistically-oriented natural sciences of the last half of the 19th century and manifested itself in part in the naïve version of the germ theory of disease that characterized the biology and pathology of this period (12). The restricted

definition of the field of etiological pathology, which Morstatt and Whetzel accept, ignores the very important principle that factors within the plant itself are part of the complexes of antecedents out of which pathic events arise, and, in being concerned only with external factors, inevitably deflects attention from the pathic plant itself.

A thoroughgoing etiological pathology considers *all* possible antecedents and recognizes that causal complexes usually include both *internal* and *external* factors. An etiology that recognizes that internal factors are part of these complexes is bound to treat the pathic plant and its processes as the central object of interest and analysis and to give experimental phytopathology a genetical, physiological, and biochemical turn. Precise etiological analysis of any complex pathic event, such as an infectious disease or a pathic form anomaly, compels one to cut the complex event conceptually and experimentally into analytical temporal and spatial isolates and to deal *seriatim* with each isolate and its antecedents. By arranging these isolates in their proper sequence, a more or less complete etiological description is given of the total pathic event. Such analysis, if rigorous, in case of multicellular organisms, invariably leads to consideration of groups of cells or of single cells and of the antecedents of their structures and processes. In other words, a full etiological description is the same as a thoroughgoing narrative of the causal pathogenesis of a pathic event and treats the structures and processes of the affected plant as the center of interest and analysis. Because of its emphasis on cellular processes of the pathic organism, a comprehensive etiology furnishes a unifying basis not only for a general phytopathology but for a comparative pathology, as well (12).

A comprehensive etiology also serves better than anything else to integrate all external and internal factors, including the effects of pathogenic foreign organisms, into a *single etiological scheme* or formula (12). At present most American and English textbooks and special articles treat predisposing factors and the factors of varietal susceptibility as categories coordinate with the category of etiology and not as integral parts of etiology. Consequently, the concepts and data arising out of genetical analysis of susceptibility and out of the experimental study of predisposing factors are not integrated in a single etiological scheme. German phytopathological literature has formally recognized that etiology deals with causal complexes and that a causal complex usually includes conditioning factors (*Bedingungen*) and an incitant (*Erreger*). It is highly desirable that American phytopathologists adopt the term "incitant" or some better equivalent to replace "the cause."

A thoroughgoing etiology, recognizing the rôle of both internal and external factors, will restore constitutional pathology as the keystone of

etiological pathology. With characteristic insight the Greek natural philosophers treated it as such, and from the time of Theophrastus (370–286 B. C.) to publication in 1853 of de Bary's first paper on pathogenic fungi (1), constitution and disposition were key concepts of plant physiology and pathology. Even during the dominance of the germ theory, Sorauer (20) and Ward (24) contended that a genuine etiology must consider the effect of predisposing factors (which they considered to be mainly external ones) upon the disposition of the plant as this expresses itself in normal and abnormal susceptibility to incitants. Latterly, Morstatt (14), in his discussion of physiological pathology, Fischer and Gäumann (5), in their discussion of the prerequisites of infection, and I (12), in a discussion of the rôle of genetics and developmental physiology in etiological pathology, have formally treated constitution and disposition as phytopathological concepts.

Finally, a comprehensive etiology also may serve to integrate experimental phytopathology and the other experimental biological sciences. In fact, the only difference between etiological pathology and each of the other experimental biological sciences is that, while these deal with the relations of the structures and processes characterizing maintenance, development, or reproduction within the range of easy tolerance of the living system in question, their pathological counterparts deal with the relations of those structures and processes that characterize difficulty or failure in maintenance, development, or reproduction (12).

A radically experimental etiology is not content to establish merely that a particular strain of *Puccinia graminis tritici* incites the pathic complex called rust in a particular strain of wheat. It also aims to account for the occurrence of the pathic event under particular past and present external conditions within a particular cell in a particular tissue of a particular age within a particular organ of a particular individual. Furthermore, it aims to explain how the presence of a particular organ of a specific parasite, which is in a particular phase of its life cycle and has a particular genetic constitution and a particular environmental past, incites this particular cellular event.

In order to attain these goals etiological phytopathology must come to grips with the problems of development and must utilize the concepts and techniques not only of the other botanical sciences, but of the nonbotanical sciences as well.

GENERAL ETIOLOGY

Experimental analysis of healthy and pathic events has shown that in addition to the obviously pathogenic external factors every factor that plays a rôle in development and maintenance and reproduction of living systems is a potential pathogenic factor (12). In other words, there is no

special class of pathogenic factors any more than there is a special class of pathic processes. Analysis has also shown that any one of these factors may play a determinative, direct or a factorial, indirect pathogenic rôle. It may act as a releasing, inciting, proximal or as a conditioning factor.

It also has been established experimentally that one factor rarely is so determinative that no other factor plays a rôle. However, such cases do occur. Lethal genes and in some cases sublethal genes are so determinative that other factors within and external to the individual are of practically no consequence. At the other extreme overwhelming external forces often play such a sudden and completely catastrophic rôle that other factors are practically negligible. In most cases, however, the antecedent of the pathic event is a complex of factors, a *causal complex* (13). Many internal and external factors cooperate in the production of an antecedent complex, which manifests itself as a *disposition* on the part of the plant to react pathically to a particular inciting internal or external factor.

The factors of a pathogenic complex may therefore be roughly divided into (a) *Internal, Constitutional Factors,* which manifest themselves in part in the *disposition* of the organization, and (b) *External Factors.*

Internal or Constitutional Pathogenic Factors. The concept "constitution" has had various meanings since its introduction into pathology by the Greek physicians and natural philosophers. Constitution, as used here, means not only the constituent parts, that is, the structures and processes of the organization of a particular individual, but also the individually characteristic manner in which these are spatially and temporally coordinated and integrated to produce the particular organization and behavior or reaction of the individual at any given instant. Sometimes the term constitution is used to designate the sum total of the possible reactions of an organism. The constitution of an organism may be analyzed into (a) its genetic, idio- or genotypic constitution, and (b) its nongenetic, para- or, less correctly, its phenotypic constitution. These do not exist as independent entities. The constitution of an organism as we see and deal with it always is the phenotypic expression or resultant of an interplay of the genetic, the nongenetic internal, and the external factors of the organism.

Genetic Constitution. By genetic constitution is meant not only the comparatively invariant part that an individual received from its parent or parents, that is, its chromosomal and cytoplasmic genes, but also the individually characteristic manner in which these are spatially and temporally coordinated and integrated into a genic system. If the contentions of Correns and Wettstein are correct, that a cytoplasmic genetic system exists in cells, then the genetic constitution of an organism, in addition to its genic constitution, also consists of a cytoplasmic part, the plasmone, and of the relation of the genic and extragenic genetic systems (12).

The direct determinative pathogenic rôle of the genetic constitution of plants is illustrated by the lethal and sublethal effects of certain chromosomal and cytoplasmic genes reported for a considerable number of plants (12). It is also illustrated by the gene effects recognized as sterility and semi-sterility and by many cases of lethal polyploidy.

The indirect or conditioning pathogenic rôle of the genetic constitution is illustrated by innumerable cases of genically conditioned susceptibility of plants to the pathogenic influence of both nonliving and living external incitants, especially of pathogenic foreign organisms.

The phase of etiological pathology that deals with the effects of the genetic constitution of the plant brings experimental pathology into intimate contact with physiological genetics. In analyzing how genes play a direct and/or indirect rôle as pathogenic factors, etiological pathology will find itself compelled to consider the propositions and concepts of physiological genetics (12). It may conceive the gene as a self-synthesizing, self-duplicating catalytic agent, which, in its primary rôle, is the antecedent of synthesis of particular enzymes and/or antienzymes at definite moments in the life history of the organization of which each gene is a part. The enzymes of each gene may be assumed to react with and upon the living substrate and/or such other materials as are present. These enzymes, in turn, in cooperation with the resultants of the action of all other genes, may be considered antecedents of the processes and structures of the cell or acellular entity under analysis. Through the intermediacy of these cells and their products, in interaction with other cells and their products, in case a multicellular organism is involved, the structures and processes of tissues and organs, and even the behavior of the organism as a whole may be considered to be conditioned by the genes. This is essentially the interpretation presented by my colleague, Sewall Wright.

Past-History and Correlative Influences—The nongenetic constitution of an organism is the relatively variable and transitory part of its organization. In the beginning the nongenetic constitution of each individual consists of the nongenetic cytoplasm and the cytoplasmic elaboration products it received from its parent or parents. Later, it consists of new cytoplasms, synthesized in the course of growth and development through interplay of the original nongenetic cytoplasm and of the genetic constitution of the individual as well as of the elaboration products of these cytoplasms. The nongenetic constitution of an organism changes in the course of the life history of the individual as differentiation of cells, tissues, and organs proceeds. It is the repository of all past events in the organism, excepting mutations in the genetic system. It learns by experience, it ages, and eventually it dies and disintegrates unless all or part of it is carried on in the formation of new individuals.

The factor of the past experience or development of the nongenetic cytoplasmic system and its products may be designated as the *past-history* factor (12). Through it, all past experiences and events of the organization tend to play a conditioning rôle in the events of the present moment of an organism. The disposition of an organism is an aspect of its past history. It is assumed that the genetic and nongenetic constitutions of a cell are in constant interplay and that the procession of events whose possibility is determined by a relatively small number of genes is released by correlative influences between genes and changing cytoplasm. It is assumed that this factor is a very important one, making it possible for a limited set of genes to determine all of the potentialities of an organism. Interplay with a changing cytoplasm may enable the same set of unchanging genes to condition new structure and behavior from moment to moment. Examples of the pathogenic influence of the past history factor are seen in those processes of differentiation that lead to death of cells, in the effect called aging, in the metabolic drift of various levels of organization of plants, and in acquired susceptibility to pathogenic agents or factors.

Mutual effects between different parts play a rôle in the constitution of every cell. Similarly, the effects of one cell and its products upon another cell in the same organism constitute a factor that conditions the constitution of the organism. These factors, internal to the organism as a whole, but external to a given call, tissue or organ, are considered the *internal environment* of the organism. They are designated as *correlative influences* or effects (12). They may be positional, mechanical, chemical, electrical, or radiative. They play a leading rôle in conditioning that the progeny of one cell (the zygote) or of a group of cells, all with the *same* genetic constitution differentiate into *diverse* tissues and organs.

The rôle of correlative pathogenic factors is seen in chlorophyll deficiencies due to incompatible nuclear and cytoplasmic constituents; in death of the zygote due to incompatible nucleus and cytoplasm; and in abscission of buds and young fruits following the ill effects of correlative transpiration in which water is withdrawn from the tender tissues by more mature and actively transpiring leaves. It is also seen in the effects of the pathic root system of the peach tree upon its aerial parts in the phony disease of the peach.

Consideration by etiological pathology of the past-history factor and of the factor of correlative influences brings experimental pathology into intimate contact with physiological genetics as well as with developmental physiology and morphology.

Disposition. The constitution of an organism may be evaluated with reference to its capacity or probability of reacting, either adaptively and favorably, or nonadaptively and unfavorably, to a given internal or exter-

nal stimulus, *i.e.*, with reference to its chances of persistence or resistance. The constitution of an individual is such that it is either capable or incapable of being stimulated into pathic reactions by a particular factor or agent. This factor thereby becomes either a pathogenic or a nonpathogenic factor as measured by the reaction of the plant. The plant, organ, tissue, or cell is said to be susceptible or resistant to or immune from pathogenic stimulation by that particular factor or, less correctly, the particular disease incited by that factor. Susceptibility and resistance or immunity, therefore, are relative polar concepts; the greater the one, the less the other.

This relative susceptibility and resistance to pathogenic stimulation are designated as positive and negative disposition (18). The term disposition, which represents a physiological concept and not a material entity, is derived from *disponere*, to be made ready or prepared.[3] When not otherwise qualified, the term disposition usually means *positive disposition* or susceptibility. In evaluating susceptibility and resistance, the usual behavior of the organization in question (species, individual, organ, tissue, or cell), with reference to its usual internal or external factors, is often taken as the norm. Disposition, consequently, is often defined as an exaggerated or abnormal reaction to stimuli.

In so far as the disposition of an organism is genetically determined or conditioned it may be said to be a hereditary, geno- or idiotypic disposition. This manifests itself especially in the differential susceptibility and resistance of different genotypes and in varietal, species, generic, and familial dispositions. It is exploited practically in selection and breeding for "disease resistance." The expression of disposition which is conditioned by nongenetic factors may be designated as nonhereditary, pheno- or paratypic disposition. It manifests itself in the individual dispositions elicited by particular environmental factors and in dispositions correlated with age, sex, organs, and external factors. An absolutely sharp line cannot be drawn between genotypic and phenotypic disposition because the actual or phenotypic disposition of an organization is always a resultant of an interplay of its genetic and of its nongenetic internal and external factors. Sometimes the genetic constitution plays such a determinative rôle that a disposition is essentially genotypic. Disposition represents the particular manner in which an organism reacts to any given stimulus at any given moment. It is the result of an integration and summation of the effects of all factors that have influenced the organism previous to that moment. Essentially, it is the past-history effect.

[3] The term diathesis (διάθεσις), a preparation, arrangement, has essentially the same meaning as disposition. Sometimes it is restricted to those dispositions that are primarily due to the chemico-physical organization of the tissues.

Sorauer (20) designated as *predisposition* the expression of unusual susceptibility by individuals of the same variety in response to environmental factors. Morstatt (13) designates individual phenotypic susceptibility as predisposition and reserves the term disposition for genotypically determined susceptibility. Fischer and Gäumann (5) use the term susceptibility (*Empfänglichkeit*) for both genotypically and environmentally conditioned individual susceptibilities, but restrict the term disposition to the latter.

External Pathogenic Factors. The class of external pathogenic factors or agents comprises all pathogenic factors that influence a living organization from without. They may be effective either singly or in combination and directly or indirectly. Every type of environmental factor is potentially pathogenic for some organization at some place, at some time. Under certain conditions of intensity, duration, and even of occurrence or nonoccurrence, a given factor may be antecedent to a healthy reaction; under others, to a pathic reaction.

Nonliving External Agents. There is no need to discuss here the various agents that comprise the categories of physical and chemical pathogenic factors. Plant pathology and, to a greater extent, certain phases of physiology and ecology, have devoted themselves to study of this phase of etiological phytopathology. By wholehearted consideration of the rôle of these factors as conditioning agents of susceptibility to pathogenic infection and as direct determinative pathogenic factors, experimental pathology comes into intimate contact with physiology and ecology.

Living External Agents. If one of the significant external factors or agents is another organism, as in predatism, pathogenic epiphytism, pathogenic parasitism, and pathogenic infection, the pathogenic activity of the foreign organism must be analyzed into its determinative and/or its conditioning factors. The attacking organism itself is an event like an attacked one, and the interplay of the stimuli and reactions of both of these organisms constitutes the most complex type of event with which the pathologist has to deal.

The relative capacity of a predator to attack, *i.e.*, its predativity, and of a foreign organism to infect or to infest, *i.e.*, its infectivity or its infestivity, may be included in the general concept "*aggressivity.*" The relative capacity to incite a pathic event may be designated as *pathogenicity* or specifically in cases of microorganisms as *virulence*. Aggressivity and pathogenicity may be considered an aspect of the disposition of an organism and are measured by the extent to which it exploits the susceptibility or overcomes the resistance of the organization with which it becomes associated. Susceptibility and resistance, on the one hand, and aggressivity and pathogenicity, on the other hand, are reciprocal or polar values or terms.

Aggressivity and pathogenicity at any given time and place are determined and/or conditioned for a given foreign organism or its parts by (a) its internal factors, *i.e.*, factors of its own constitution and (b) its external factors, of which the constitution or disposition of the attacked plant, by virtue of its susceptibility or resistance, is the most important one. At times, factors external to both organisms affect the foreign organism directly. The internal factors for the foreign organism are its genetic constitution, its past-history factor, and the factor of correlative influences.

If the genetic constitution is the sole or determinative factor in aggressivity and pathogenicity these may be designated as hereditary, genotypic, or idiotypic aggressivity and pathogenicity. The actual phenotypic aggressivity and pathogenicity manifested at a given time and place, however, are resultants of an interplay of internal genetic and nongenetic factors and of external factors. Fischer and Gäumann (5) distinguished between (a) innate, constitutional differences in aggressivity and (b) changes in aggressivity, which they call disposition. As pointed out previously, it seems preferable to use this term to represent the phenotypic expression of the resultant interplay of internal and external factors. The aggressivity and pathogenicity of an organism may be correlated with age, sex, taxonomic relationship, nuclear phase, developmental phase, as well as with definite organs, tissues, or cells of an individual.

The phase of etiological pathology that deals with pathogenic parasites brings experimental pathology into intimate contact with parasitology, especially with its bacteriological, mycological, nemathelminthological and entomological aspects. In so far as it deals with the internal factors that condition the aggressivity and/or pathogenicity of predators and parasites it brings experimental pathology into intimate contact with the genetics and developmental physiology of these organisms. Since the time of de Bary, etiological phytopathology has been predominantly parasitological, and parasitism and infection have been its leading concepts.

SUMMARY

For the sake of convenience the factors or agents that may condition and/or incite a pathic event within an acellular[4] or unicellular organization or within any given part of a particular tissue or cell of a multicellular organism may be summarized in the following tabular form:

I. Internal factors (factors within the organism)
  A. Genetic constitution
    1. Chromosomal genes
    2. Cytoplasmic genes
    3. Nongenic genetic factors (plasmone)?

[4] By acellular is meant a living organization not involving a nucleus and cytoplasm.

B. Past history
   1. Original nongenetic constitution
   2. Acquired nongenetic constitution
C. Correlative influences
   1. Between parts of the same cell or of an acellular organism
   2. Between different cells or tissues and/or their products

II. External factors (factors outside the organism)
A. Physical agents
   1. Light and other rays
   2. Heat
   3. Cold
   4. Electricity
   5. Radiations
   6. Mechanical-traumatic agents
B. Chemical agents
   1. Toxic substances
   2. Enzymes
   3. Hormones (growth-promoting and inhibiting substances)
   4. Nutritive and metabolic substances
C. Organized living agents
   1. Organisms effective without contact
   2. Organisms effective by temporary contact (predators)
   3. Organisms effective as epiphytes and epizoa
   4. Organisms effective as parasites (plant and animal)

The aggressivity and pathogenicity of each pathogenic foreign organism must be analyzed so far as possible into their determinative and/or conditioning factors, which are of the same kind as those listed in categories I and II. To conserve space these are not repeated for each subcategory of category II C. Schemata that diagrammatically represent the possible influences of these factors as antecedents of a given pathic event in a given place at a given moment have been presented in an earlier paper (12).

II. INFECTION AND PARASITISM

a. *Infection*

It is obvious that adoption of a thoroughgoing etiology removes infection and parasitism from the center of etiological pathology. They nevertheless are such important aspects of etiology that they deserve special consideration.

The term *infection* is derived from the Latin verb *inficere,* which means to put in, taint, pollute, defile, contaminate. In present-day biology the term has come to mean: the act of communicating a foreign organism, specifically, a microorganism; the foreign organism which is communicated; the state of harboring a foreign organism; the event of interaction between the foreign and the contaminated organisms; the pathic reaction of the contaminated organism to the presence of the foreign organism; the act of

communicating disease; a disease which is communicable. In this discussion the term infection is used to designate the *state of being contaminated* by a minute foreign organism. Typically, this contaminating organism is microscopic; at least when it lodges upon and/or enters and permeates the affected organism. At one extreme, as in contamination by filterable viruses, infection therefore grades imperceptibly into intoxication by poisons; at the other, as in the parasitism of mistletoe, it grades imperceptibly into infestation, that is, contamination by a large foreign organism.

The term *contagion* has essentially the same meanings as infection. It is derived from the Latin verb *contangere*, which means to touch. Sometimes the term *contagium* is used to designate that which is communicated, while contagion is used in contradistinction to express the act of communicating. Contagion is also defined as infection by contact.[5]

b. *Parasitism*

Many infections involve the event of parasitism. The concept parasitism has been variously and contradictorily defined. The definition here used is an adaptation of one given by Thompson (21). If an organism of one kind lives on or within a living individual of the same or of another kind, the host, in a one-sided nutritive relationship, which is not beneficial and is often harmful to the host, the individual is said to be a parasite. The concept parasitism, therefore, is formulated to apply at the individual or species level of organization of biological entities. It is, therefore, essentially an ecological concept. The organism on or within which the parasite is living and from which it derives its food is said to be parasitized. The relation of parasitism relieves the parasite from most of the activity and struggle usually associated with procuring food and thus tends to induce some degree of simplification or degeneracy. *One-sided food relationship* between two associated individuals, therefore, is the essence of parasitism. This was the original implication of the term. In Greek society a person who was assigned by social custom and law to eat at the table of another was designated as a parasite (παρασιτος). The biological concept implies more than a space relationship, but it does not necessarily imply a biologically significant harmful effect, even though it precludes a mutually beneficial relation.

The first detailed discussion of parasitism of plants is found in the "Causes of Plants" by Theophrastus (27). In his analysis of the relation of mistletoe to trees he recognizes the mistletoe as a separate, independently propagating plant whose seeds are disseminated by birds. Theophrastus

[5] The history of these concepts will be published as a separate paper, "Pathic Events in Plants and the History of Infection."

recognized that the mistletoe lives at the expense of the infected tree and that this association often leads to harmful effects for the tree. In this first phytopathological classic on parasitism Theophrastus does not transfer the term parasite from the social to the botanical vocabulary. It is significant that he does not discuss the mistletoe in the chapters devoted to anomalies, injuries, diseases, and death of plants, but in the course of his discourse on the nutrition of trees. He compared its nutritive relation to its host with that of a scion to its stock. He did not, of course, know that the green leaves synthesize part of the food of the scion. Theophrastus also recognized that dodder, orobanche, and fleshy fungi live attached to and at the expense of other plants, often with fatally pathogenic effects.

Tubeuf (23), in his excellent monograph on the mistletoe, lists examples of superstitious beliefs and folk and medical lore of various peoples of Europe about this interesting plant. These show clearly that the anomalous mode of life of the plant rendered it an object of early and special interest to prescientific man. The mistletoe is listed in the earliest herbals of Europe. Matthioli (1586) considers it a self-propagating plant. Later (1679), Malphigi studied the anatomy of the mistletoe and considered it a parasite that incites galls. He does not, however, designate it by the term parasite. The mistletoe apparently was not called parasite in the scientific literature (although early German literature designates it Schmarotzer) until the 17th century.

Sarauw (19) states that Micheli (1729) introduced the term parasite into scientific literature in connection with his discussion of the Balanophoraceae. Micheli, in fact, first used the term parasite in 1720. The first record I have been able to find of its use in botanical literature is by Ray (16) in his Historia Plantarum of 1688. The close of the general discussion of the first volume (1670) of this significant treatise is devoted to a thoroughgoing naturalistic discussion of Diseases of Plants. In discussing the mistletoe, Ray says "Viscus est planta parasitica."[6] Ray, quoted by Duhamel (3), also reports that the London apothecary Doody succeeded in growing the mistletoe from seed and thus demonstrated experimentally that it is an independent self-propagating plant. Tournefort (22), in his Memoir on Diseases of Plants of 1705, in the course of a discussion of the external incitants of disease, including fungi, designates a considerable assemblage of plants, including dodder and mistletoe as parasitic plants.

The first record of unequivocal application of the term parasite to a definite fungus that I have been able to find occurs in a memoir by Duhamel (2) in 1728 on the decay of Safron. In this phytopathological

[6] A reference by Guettard (8) to Morison's Plantarum Historia Universalis Oxoniensis may indicate that Morison may have applied the term parasite to certain plants in 1672 or 1680.

classic the term parasite is applied to a microfungus, which Duhamel considered a truffle-like plant, now designated as *Rhizoctonia crocorum*. Duhamel proved experimentally that this fungus is able to live in the soil; that it may grow into and live at the expense of the safron bulb; that it may incite decay of the scales with attendant death of the entire plant. He showed that it could parasitize other Liliaceae and Amaryllidaceae. He suggests isolation, rotation, and sanitation as control measures. This paper, which is a landmark in parasitological mycology and plant pathology brought Duhamel election to the Royal Academy. The fact that Tournefort (1705) and Duhamel (1728) use the verb parasitize, the adjective parasitic, the noun parasite, without the type of discussion usually attending introduction of new concepts and terms, may indicate that these terms had been used previously in French scientific literature. Tournefort merely says, after listing dodder, mistletoe, and other plants, "With good reason these are called parasitic plants." The term parasite was part of early literary French and English. Rabelais (1495–1533), Shakespeare (1564–1616), and Ben Jonson (1573–1637) applied it to certain characters of their plays.

Guettard (8), in a memoir on the adherence of dodder to other plants (1744) and (9), in a memoir on parasitic plants (1746), recognized levels of parasitism ranging from slight to full food dependence of the parasite on the host. In 1762 Duhamel (4) devoted Book V, Chap. I, Art. V, of "La Physique des Arbres" to a discussion of "Des plantes parasites." By 1767 Fontana (6) recognized rusts of wheat as parasitic, self-propagating pathogenic fungi.

It is contended in some quarters that *Rhizopus nigricans*, which often grows on or within the storage root of the sweet potato, and derives its food from this organ, or *Fomes pinicola*, which often grows within and derives its food from the dead heart-wood of living trees, should not be regarded as parasites because they invade only through wounds and derive their food only from dead cells. This interpretation of the concept of parasitism has led to much confusion. It is based in part upon a failure to recognize that a concept formulated to apply to an organism as an individual or whole is not necessarily applicable to its tissue or cellular level of organization. The essence of the concept of parasitism, in so far as nutrition is concerned, is dependence of *one individual* upon another living individual for food, irrespective of whether the materials used by the dependent individual or parasite are actually derived from the living or dead parts of the host. Consequently, a heterotrophic organism such as *Rhizopus nigricans* or *Fomes pinicola* may be considered a parasite in that it obtains its food from

a living individual[7] even though it actually uses only nonliving constituents of the host.

It is convenient, therefore, to recognize different food habits in parasites. One group of parasites derives its food from the living tissue of the host, *i.e.*, from living cells and/or their immediate products, such as the cell walls. The other group, while living on and at the expense of their hosts, derive their food from dead tissues and cells and their products. The first group may be said to be living biotrophically (*βιος*, bios = living; *τροφή* trophe = food), or as biotrophic parasites. The second group of parasites may be said to be living necrotrophically (*νεκρός*, necros = dead) or as necrotrophic parasites. If one prefers, with Münch (15), to distinguish between those necrotrophic parasites that kill tissues before they use them as food and those that utilize as food the tissues killed by agents other than themselves, one may say that the former carry on nutrition thymatrophically, or as thymatrophic parasites (*θῦμα*, thyma = victim), and the latter saprotrophically or as saprotrophic parasites (*σαπρός*, sapros = rotten). The latter term is applicable only because in biology sapros has come to mean more than rotten, being used in the sense of nonliving. Thus an organism growing on a dead body or on an artificial medium is currently designated as a saprophyte. The first group includes all of those pathogenic parasites that kill and destroy tissues by means of toxic by-products and enzymes prior to their invasion and utilization as food. An organism living as a parasite may, therefore, obtain its nutrients either biotrophically or necrotrophically. These terms recognize the differences that exist between *Puccinia graminis*, which lives on the living tissues of wheat, and *Fomes pinicola*, which lives on the dead heart wood of a standing, living tree. In the latter case the presence of the parasite has potential pathogenic implications for the tree as a living whole, even though, considered at the cellular level of the host, the parasite is living only at the expense of the dead tissues.

The concept infection, therefore, is a broader one than parasitism. All cases of parasitism are infections or infestations but not all infections involve parasitism. Thus the epiphytism of microorganisms is a phase of infection but not of parasitism. Furthermore, not all parasites are pathogenic; hence, one must distinguish between pathogenic and nonpathogenic parasites. The flagellate *Herpetomonas* spp., which live in the latex of *Euphorbia* spp. are nonpathogenic parasites.

If an infection is part of the antecedent complex of a pathic event it is a *pathogenic infection* and the resultant is designated as an *infectious disease*.

[7] Because of the low level of organization of plants certain plant organs such as a sweet-potato storage root and other parts of plants capable of continued existence when severed from the plant as a whole may be considered individuals.

This use of the term infectious disease differs from the usual meaning of the term that emphasizes communicability or capability to communicate. In fact, common usage with its reflection of the ancient anthropomorphic notion that diseases are discrete entities is incorrect in the light of biological conceptions about the nature of pathic events. In reality, as pointed out by Henle in 1840 (10), only the infective pathogenic agent, *i.e.*, the incitant, is communicable; the pathic processes, the essence of the pathic event, are not transmissible.[8] If the infective agent is transmissible by contact alone the resultant pathic event is often designated as a contagious disease in contradistinction to infectious diseases, in which the infective principle may be communicated by carriers such as other organisms, soil, water, and air. In common usage, however, the terms contagious and infectious diseases frequently have essentially the same meaning.

Obviously not all infections are pathogenic. Many individuals of the heath family harbor various mycorrhizal fungi (17), *i.e.*, they are infected. However, some of these infected plants, as individuals, do not display any pathic reactions: in fact, the association is supposed to be of actual benefit to them. Nevertheless, particular infected cells may be pathic in that they degenerate and die prematurely because of the infection. Here again, it must be borne in mind that an infection may be nonpathogenic when evaluated with reference to an organism as a whole but pathogenic when evaluated with reference to particular organs, tissues, or cells of this individual. Thus the legume plant as a whole may be benefited by an infection of nodule-inciting bacteria, whereas particular cells in the root are necrotized or incited to pathic development and reproduction. These and the wound responses of many other cells not invaded, which often differentiate into new kinds of tissues, give rise to the gall. All gradations of harmful and beneficial effects to the plant as a whole are exhibited by nodule-bearing legumes. It appears that the bacteria are not able to readily invade the roots or establish themselves unless a soil-nitrogen deficiency so seriously disturbs the carbohydrate-nitrogen metabolism of the plant that it may be considered pathic. A benefited infected plant may, therefore, be considered as a plant that was pathic that became locally infected as a consequence but that, by virtue of the secondary effects of the local infection, overcame the original pathic state. Additional data are found in a monograph by Fred and associates (7).

In infectious diseases, as well as in diseases incited by the larger foreign organisms, the incitant releases chains of reaction or initiates processes in the infected or infested plant that in turn set up stimulus-reaction chains in the infective or infesting agent. These reactions manifest themselves in

[8] It is possible that some chlorophyll deficiencies involve transmission of a pathic cytoplasm from mother to progeny through the megagamete.

part in the susceptibility and resistance of the infected plant and in the infectivity or invasiveness and virulence of the infective agent. The resultant is the most complex type of pathic event found in living systems. It consists of process complexes that manifest themselves in regressive, necrotic, repressive, and/or progressive pathic changes in both the infecting and infected organism. These process complexes consist of processes that differ in no way from those that characterize pathic events whose etiological complex does not include an infective agent or from those that characterize healthy events. They differ from those of healthy events largely in that the duration, intensity, and combination of the constituent processes are characterized by lack of regulation and integration.

III. ETIOLOGICAL CLASSIFICATION OF PATHIC EVENTS

Beginning with the taxonomic phase, which dominated biology during the 18th century, attempts have been made to classify pathic events as though they were discrete entities. Pathic events do not exist apart from individual living entities and hence can be classified only if they are abstracted into concepts that generalize the particular events occurring within individual organisms. Such abstraction, however, is necessary if the maze of our sense experiences about pathic events is to be coordinated into a convenient system and if relations are to be formulated and a scientific presentation made possible.

Etiology concerns itself in part with the classification of antecedents of pathic events, but it is not concerned with the classification of pathic events themselves. Etiology, however, has furnished several bases for classifying pathic events that have proved more convenient than systems based on other criteria. Consequently etiological classifications of pathic events have become and probably will remain dominant in phytopathology.

In consonance with the classification of pathogenic factors previously given, pathic events may be grouped as follows:

I. Pathic events incited or determined by internal factors
   A. By genetic factors
   B. By nongenetic factors.
II. Pathic events incited or determined by external factors
   A. By physical agents
   B. By chemical agents
   C. By organized agents
      1. By organisms effective at a distance
      2. By organisms effective by temporary contact (predators)
      3. By organisms effective as epiphytes and epizoa
      4. By organisms effective as parasites.

In this classification the phrase "pathic event incited or determined by" is to be read to mean a *pathic event in whose etiological complex* a particular factor plays the inciting or determinative rôle.

Pathic events are grouped most frequently as nonparasitic and parasitic pathic events (diseases). This system or variants of it have long been in vogue and are widely used today. In the classification just given, all categories, excepting II 4, represent non-parasitic pathic events. If "parasitic disease" is defined to mean any pathic event whose etiological complex includes a parasite, *i.e.*, as a pathic event that is incited or determined by a parasite, and "nonparasitic disease" is defined to mean any pathic event whose etiological complex does not include a parasite, i.e., as a pathic event that is not incited or determined by a parasite, these categories are etiologically sound and their use does not necessarily stifle the development of general cellular phytopathology nor split the field of phytopathology into applied botany and applied zoology. This results only if the *content or subject-matter* of phytopathology is delimited on an etiological basis and is avoided if the essence of the concept "pathic event" is taken to be a *harmful occurrence* within an organism rather than an occurrence brought about by a *particular kind* of factor or agent, or by a factor that is not a *usual part* of the environment of the affected organism (12).

IV. ETIOLOGICAL CLASSIFICATION OF CONTROL MEASURES

An etiological classification of pathic events that recognizes that factors both within and without the plant may play inciting and or conditioning pathogenic rôles gives a basis for classification of control measures that is of use in general plant pathology. Control measures, on this basis, may be placed into one category that comprises those measures that are directed primarily or solely at the potentially or actually pathic plant as a pathogenic factor and into a second category that comprises those directed primarily or solely at factors outside the potentially or actually pathic plant, as pathogenic factors.

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CERTAIN SCLEROTIUM DISEASES OF GRAINS AND GRASSES[1]

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INTRODUCTION

A fungous disease, which appeared in the wheat fields when snow was melting, was first reported in Idaho (4) in the spring of 1922 in Fremont and Teton Counties. This disease has occurred in this region with more or less severity each year since then. It was of considerable importance in 1931 and 1932 on the University of Idaho Substation Farm at Sandpoint, Idaho, and was reported in 1931 from near Hill City, Idaho, and in 1932 from near Cascade, in Long Valley, Idaho. This same disease has been reported on wheat from Washington[2] and Montana[3,4] and has been found on lawn grass in Ithaca, New York,[5] and at Moscow, Idaho. Injury from apparently the same disease is noticed almost every year in northern parts of Japan (1, 5). It has been found in northern Germany (3, 6, 7) and in the Scandinavian countries (2).

The disease is most commonly found at high altitudes where winter wheat is grown and in localities where the snow disappears late in the spring.

DESCRIPTION OF DISEASE

The disease was first attributed to snow injury but later a fungus was found always associated with it. The plants seem to be infected under the snow, since portions of the leaves appear scalded and discolored in spots when the covering of snow is taken away. After the snow melts, these leaves die and become greyish white, on drying, and finally become matted together on the ground. When a field is heavily damaged, entire areas are covered with greyish white dead plants. When the damage is slight, green leaves may be seen here and there. As the weather becomes warmer, the activity of the fungus ceases, and the partially infected plants grow vigorously, and often the effects of the disease are gradually overcome. Because of its gen-

[1] Published with the approval of the Director as Research Paper No. 101 of the Idaho Agricultural Experiment Station.

[2] Heald, F. D. *Sclerotium rhizodes* Avers. U. S. Dept. Agr. Bur. Pl. Indus. Pl. Dis. Rptr. Suppl. 35: 270. 1924.

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[4] Young, P. A., and H. E. Morris. Sclerotium root rot (*Typhula graminum* Karst.). U. S. Dept. Agr. Bur. Pl. Indus. Pl. Dis. Rptr. Suppl. 69: 127. 1929.

[5] Dr. H. H. Whetzel of Cornell University kindly sent the writers a specimen of lawn grass on which were found a few sclerotia. A culture was secured from these sclerotia.

eral appearance in the field, it has been called "snow-mold" and "snow-rot" by Japanese workers (1, 14, 15). Due to the scalded appearance of the plants as the snow melts, the writers suggest the name "snow scald."

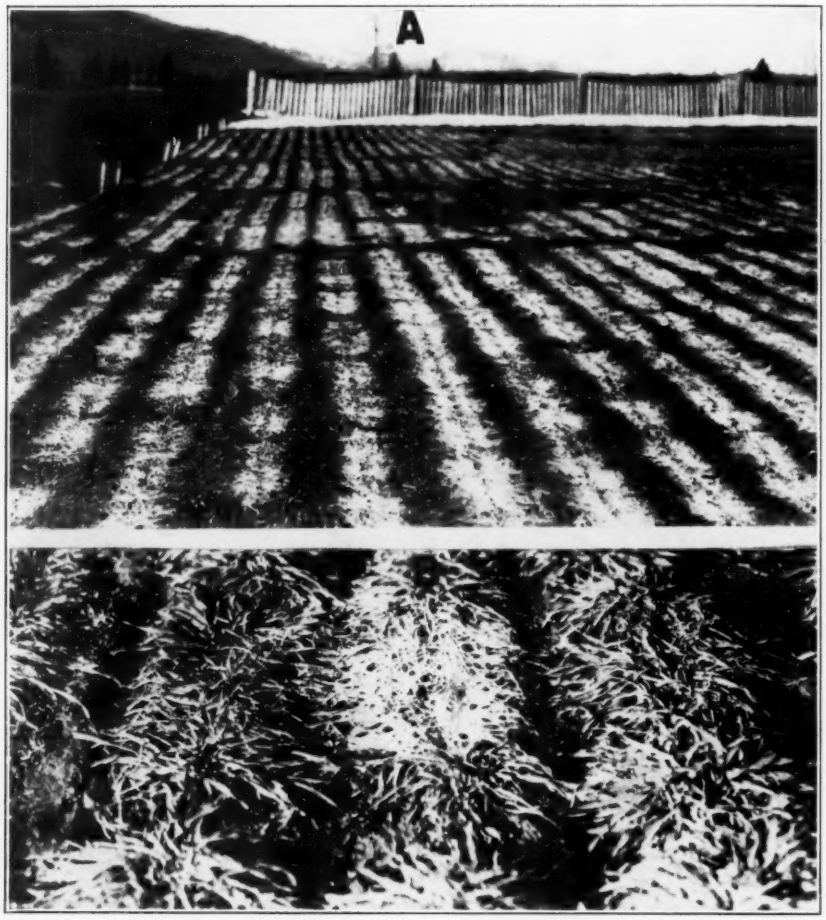

FIG. 1. A. Winter cereal plots on the University of Idaho Substation Farm at Sandpoint, Ida. These plots were almost entirely destroyed by the snow-scald fungus. B. Healthy and diseased Mosida winter wheat plants soon after the snow had melted. Note that only a portion of one plant was diseased. Photographs by J. H. Christ.

In the first stages of the disease, the infected parts are covered with a white growth and, later, brown sclerotia appear in abundance throughout the infected tissue. After the snow disappears, layers of white, mouldy growth are found on the soil and infected plants. (Fig. 1, A.) Soon the infected leaves lose their green color, become dry, and lie matted on the

ground. Some of the inner leaves may still be green and the plant only partly killed. The leaves, stems, crowns, and roots of the plants may show signs of infection. The roots of the infected plants are often found rotted, and mycelia appear on them near the surface of the ground. Often the leaves, stems, and other infected parts are covered with sclerotia that

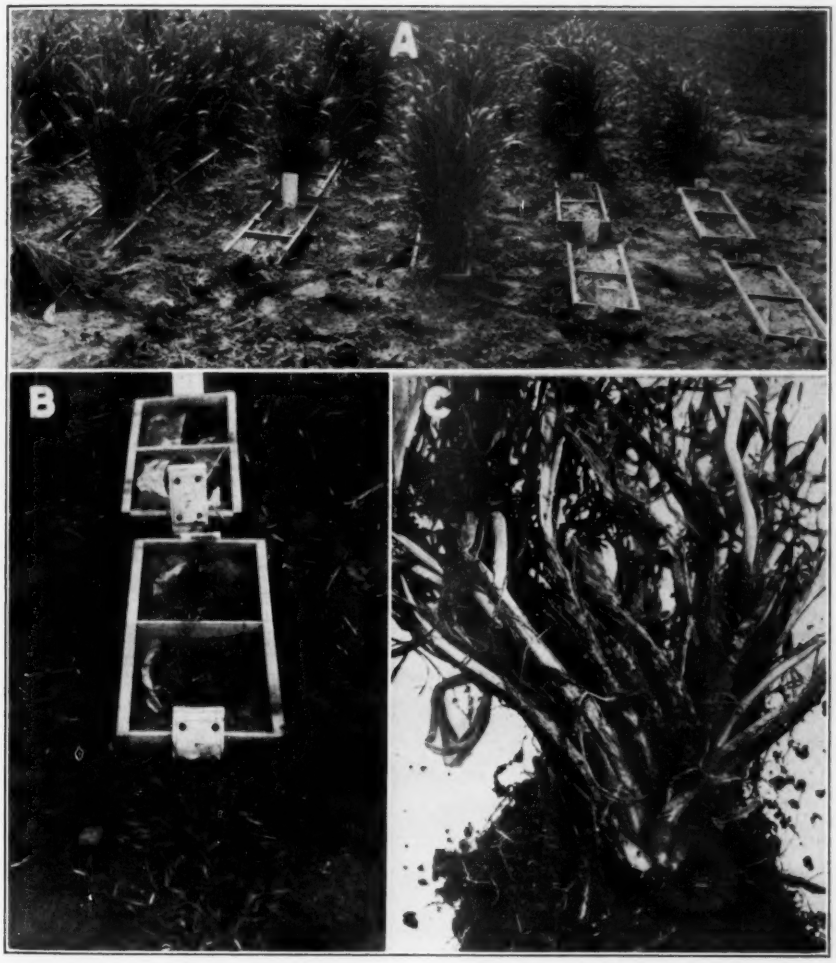

FIG. 2. A. and B. Wooden frames used to test the pathogenicity of various cultures of fungi. Cultures containing sclerotia were added to the soil inside the wooden frames in the spring. The frames were then covered with cloth during the summer. Wheat was planted Sept. 25 in the frames and in the noninoculated soil adjacent as a check. Note that nearly all plants were killed in the frames. C. Infected winter wheat plant taken from the field early in April. Note the numerous sclerotia embedded in the tissues.

range in size from an ordinary pinhead to several times as large. (Fig. 2, C.) Early in the season the sclerotia are yellow or light brown but turn dark later. When mature they are ovoid to spherical, dark brown, and of irregular, rough contour. They are often massed together. They are fastened so securely to the infected plant that it is almost impossible to remove them without tearing the tissue. These sclerotia may also occur on the roots an inch or two below the crown and a great many of them are often found embedded within the leaf sheaths. They are very numerous on the leaves of the dead plants or on plants that have been partially killed. On the partly killed plants light brown, spotted areas appear along the inner leaves, while the outer areas die. The fact that portions of the plant may be attacked or killed by the fungus while the rest of the plant is apparently healthy, has been considered good field evidence of the pathogenicity of the fungus. (Fig. 1, B.) During the summer, these plants, dwarfed and weakened by the disease, are found scattered over or along the edges of the infected areas. Only a few short heads develop upon such plants and the kernels of wheat produced are badly shrivelled. The dead areas in the field correspond to places where the snow disappeared late in the spring. Since the plants become infected during the winter, it is thought that the sclerotia, which fall to the surface of the ground, or remain on the stubble in the field, may be the source of infection the following year.

ECONOMIC IMPORTANCE

The disease ranges in severity from a slight trace to a total loss. In high, dry fields the damage is only slight, while in low, moist places or where the snow remains on the field late in the spring, the damage is very severe. In the fields in Teton and Fremont Counties, in 1923, the damage ranged from a trace to 50 per cent. At the Sandpoint Substation in the experimental plots of wheat and barley, in the spring of 1931, the damage ranged from 5 to 85 per cent. In some cases the yield was reduced 75 per cent. When the disease is very severe, the fields often have to be reseeded to spring wheat. In 1922, 1926 1929, and 1932 snow scald caused varying amounts of damage in certain areas of Teton and Fremont Counties. In each of these years from several hundred to several thousand acres of winter wheat were killed and had to be reseeded in the spring.

EXPERIMENTAL DATA

The causal organism may be easily isolated from either the sclerotia, which are formed so abundantly in the infected parts of the plant, or from the infected plant tissue, themselves. The fungus grows readily upon various media, and sclerotia form abundantly from 1 to 4 weeks later.

Specimens of diseased plants or cultures of organisms from grains and grasses have been obtained from 10 different sources.

1. Culture No. 226 came from material sent to the experiment station from Drummond, Idaho, (altitude approx. 6,000 ft.) in 1926.
2. Culture No. 227 was isolated from diseased wheat plants from Felt, Idaho, (altitude approx. 6,000 ft.) in 1930.
3. Culture No. 278 was obtained in 1931 from diseased barley plants from the Sandpoint-Substation variety-test plots (altitude approx. 2,127 ft.).
4. Culture No. 279 was obtained from Hill City, Idaho, (altitude approx. 5,200 ft.) in 1931 from a portion of a wheat field where snow had remained rather late in the spring.
5. Cultures No. 280 and No. 281 were isolated from 2 diseased specimens of wheat from fields at Felt, Idaho, in 1931.
6. Culture No. 282 was obtained from diseased grass from Ithaca, New York, in 1931.
7. Cultures Nos. 283, 284, 285, and 286 were obtained from Heizi Tasugi, Tokyo, Japan, in 1931. These were isolated in 1926 and 1928 from barley, wheat, timothy, and foxtail grass (*Alopecurus fulvus* L.).
8. Culture No. 287 was obtained from grass from a lawn in Moscow, Idaho, (elevation 2,600 ft.) in 1932.
9. Culture No. 288 was obtained from orchard grass (*Dactylis glomerata* L.) on the golf course in Moscow, Idaho, in 1932.
10. Culture No. 289 was isolated from diseased barley in the variety test plots at the Sandpoint Substation in 1932.

TABLE 1.—*Diameter of vegetative mycelia and size of sclerotia of the organisms in each of the 4 groups of fungi*

Group	Culture	Diameter of vegetative hyphae	Size of sclerotia
	No.	μ	*Diam. in mm.*
I	226	2.44 – 3.33	0.1 – 0.25
	277	4.22 – 4.88	0.9 – 2.0
II	279	2.89 – 4.44	0.9 – 1.2
	281	3.55 – 4.44	1.4 – 2.0
	278	2.88 – 4.44	1.0 × 1.5 – 1.9 × 2.0
	282	3.21 – 4.66	1.8 × 2.0 – 2.0 × 4.0
	283	3.55 – 4.44	2.0 × 2.3 – 2.5 × 2.5
III	284	3.77 – 4.66	1.4 × 2.0 – 2.0 × 2.2
	285	2.89 – 3.77	1.6 × 1.6 – 2.0 × 2.3
	286	2.44 – 4.22	2.0 × 3.0 – 2.5 × 2.9
	287	3.33 – 4.44	1.1 × 1.5 – 1.7 × 2.0
	288	3.55 – 4.44	1.5 × 1.9 – 1.5 × 2.0
	289	3.21 – 4.44	1.2 × 2.0 – 2.0 × 2.2
IV	280	4.44 – 6.66	4.0 × 5.0 – 6.0 × 10

These 14 sclerotia-forming fungi have been studied carefully. Observations on their growth characteristics and reactions to environmental conditions have resulted in classifying these various isolations into 4 different groups, those in each group having approximately the same characteristics.

MORPHOLOGY

When a sclerotium germinates, it sends out white, branched, septate mycelia. These mycelia at first adhere closely to the surface of the medium,

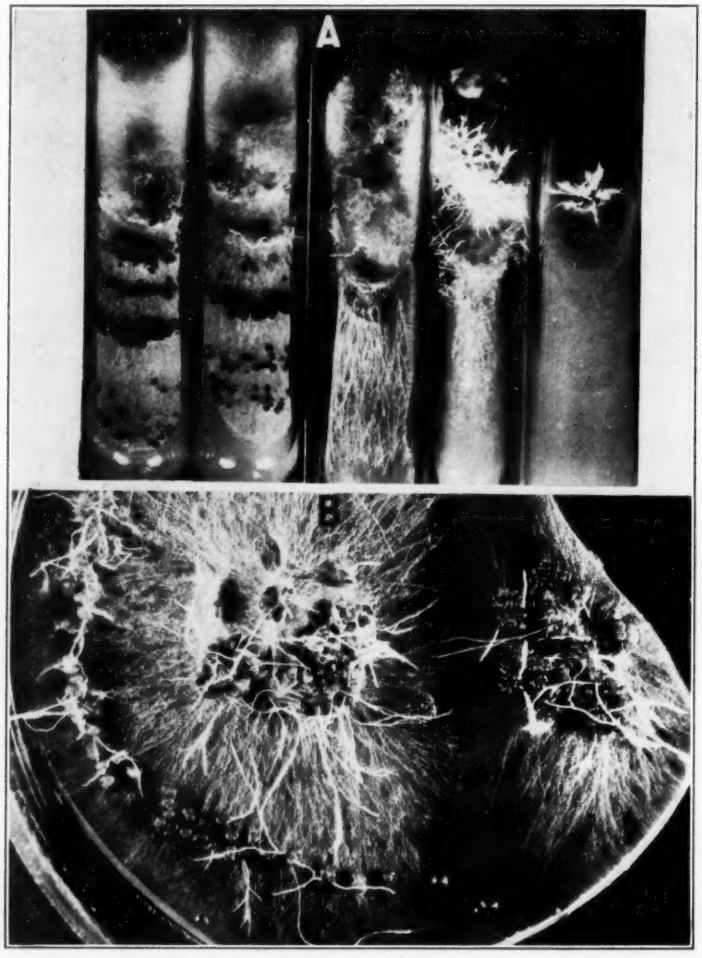

FIG 3. A. Cultures showing characteristic progressive development of sclerotia on potato-dextrose agar slants. B. 138-day-old culture No. 278 growing on potato-dextrose agar. This photograph illustrates the typical fluffy mycelia and the thread-like growths composed of compact masses of mycelia growing from the sclerotia.

but later become aerial and fluffy. The hyphal septae may be either at right angles or oblique to the long axis of the hypha. In cultures belonging to groups II and III the septae are accompanied very frequently by clamp connections. The long hyphae branch and rebranch many times. Growth is very slow in group IV as compared with the other groups. The content is more or less granular and the cells often appear vacuolated. Sclerotia are formed by a dense cluster of hyphae whose cells become somewhat enlarged. The young sclerotia seem to originate from a single hypha or from two to several knotted together. Sclerotia may form in a central mass in culture or in concentric rings or profusely scattered over the surface of the media. (Fig. 3, A.) They are often accompanied by honey-colored drops of watery secretion. In group I, the sclerotia appear both rapidly and profusely and are separate and distinct from each other. They have no rind in cross-section, are light brown to black when mature. They are spherical to oval in shape and smooth. The sclerotia in group II do not appear so rapidly as those in group I, and may appear agglomerated. They have no rind in cross-section, are spherical to irregular in shape, and either smooth or rough. They are light brown when young and dark brown when mature. Sclerotia in group III appear with about the same rapidity as in group II but often coalesce into large spongy masses. They are pinkish orange and gelatinous when young and reddish brown with a tough rind when mature. They are rounded to irregular in shape and superficially rough. The sclerotia in group IV are very slow to appear in culture and are often massed together. They are milky white when young, black when mature, and have a heavy black rind that surrounds a white medullary area in cross-section. They are large and irregular and are often cup-shape on one side.

Very often sclerotia will develop long, slender, thread-like rhizomorphs composed of compact strands of hyphae. (Fig. 3.) In cultures belonging to group II, these growths resemble brown, horn-like, simple or branched projections 5–15 mm. long. In cultures belonging to group III, they resemble long simple or branched white threads 5–50 mm. long. These same rhizomorphs develop in the field during summer and early autumn from sclerotia in the soil.

PHYSIOLOGY

Various media have been used in culturing the organisms, but potato-dextrose agar has thus far given the best vegetative growth and sclerotial development in almost every case.

The sugar content of potato dextrose was changed by adding Karo syrup in varying amounts. The cultures were grown also on media containing synthetic nutrient solutions (9) in an attempt to induce sporulation.

Culturing on acid media also was tried in an attempt to produce the perfect stage (10).

The organisms react very definitely to variations in temperature. Those in group I attained optimum growth at 10° C., those in group II at 5° C., those in group III at 0°–5° C., and that in group IV at 5° C. In all instances growth was better in the dark than when any light reached the cultures. Growth was greatly diminished at temperatures from 10° C. to 20° C., while the organisms ceased growing entirely at temperatures higher than 25° C. Because of this maximum growth of the fugus at low temperatures in culture and the occurrence of the disease associated with snow and cold weather in the field, it would appear, then, that the maximum development of the fungus is definitely favored by cold weather.[6]

Sunlight, also, seems to have a pronounced influence on the growth of the fungus. Mycelial growth is retarded and sclerotia fail to develop in cultures that have been exposed to direct sunlight. Diffused light retards growth but the effect is less pronounced than is that of direct sunlight. Tasugi (14), however, who has done extensive work with the organism in Japan, has shown that sunshine plays an important rôle in the production of basidiospores. He reports that sclerotia, exposed to sunshine while in the soil, will produce hymenophores bearing basidiospores.

In attempting to obtain the perfect stage of the fungus, cultures were exposed to both direct sunlight and to ultra-violet light. Exposures to direct sunlight were given between the hours of 11:00 A. M. and 3:00 P. M. and were of varying duration. However, no tendency toward sporulation was observed.

Stevens (11, 12, 13) and Ramsey (8) have been able to produce the perfect stages of various fungi by exposure to ultra-violet light. Cultures of sclerotia growing on nutrient media and also soil, containing sclerotia from the field, were exposed to ultra-violet light for different lengths of time at several distances from the source of light. Exposure both to sunlight and ultra-violet light were made through Cellophane, which was sealed over the dishes in order to keep the cultures sterile during the time of exposure. Cellophane was used because ultra-violet rays will pass through it, while they are screened out by ordinary glass. Some of the cultures of Nos. 278 and 285, when exposed to ultra-violet light, produced a very few growths resembling sporophores and originating from the sclerotia. A few tiny spore-like bodies were seen associated with these growths. Although the experiment has been repeated, no sporophores developed. This is the only indication of the perfect stage yet found in any of the cultures. This evi-

[6] Observations indicate that damage is more severe when the ground is unfrozen under the snow during the winter months. Doubtless the fungus is more active under those conditions than when the ground is frozen before snow falls.

dence, however, was inconclusive and until more careful study can be made of these phenomena no definite conclusions can be drawn.

Because the complete life cycles have not been worked out satisfactorily it is impossible to definitely classify the organisms. However, the cultures sent from Japan by Tasugi, which he classifies as *Typhula graminum* Karst., appear to be similar to those in group III.

PATHOGENICITY TESTS

Pathogenicity tests of the organisms on plants grown in the greenhouse were unsuccessful. This probably was due to the fact that the temperatures in the greenhouse were too high for infection to cause any material injury. Wheat was sown in sterilized soil in pots and placed in the ice chest after having been surface inoculated with sclerotia produced from culture No. 226. Browning of the leaves and stems occurred and evidence of hyphae among the tissue was present but no sclerotia formed. However, successful inoculation was obtained with Nos. 282 and 285. Sterilized kernels of Turkey Red wheat were planted in sterilized Ottawa silica sand, which was kept moist with Sach's nutrient solution throughout the experiment. One week later sclerotia from cultures 282 and 285 were placed on the sand. Large test-tubes 12 inches long, containing sand and the wheat plants, were placed in the dark in refrigerators at temperatures just above 0° C. In one location the test tubes were subjected to occasional temperatures below 0° C. so that ice formed periodically around the plants. It was in these test-tube cultures that the most successful infection was obtained. Mycelial growth occurred up the stems of the wheat plants and sclerotia were formed embedded in the tissues and along the roots. (Fig. 4.) Reisolations of the two organisms were made from both sclerotia and infected tissue. It was possible also to inoculate plants with sclerotia produced in the field. Soil containing sclerotia was obtained from infested plots at Sandpoint and allowed to remain under outdoor conditions during the summer. Turkey Red wheat was planted in some of this soil that had been brought to the laboratory in the fall. The soil was put in glass tumblers and placed in a refrigerator at a temperature of about 0° C. It was kept moist with Sach's nutrient solution. Sixty days later one plant out of three inoculated was killed; mycelial growth was profuse up the stems and over the surface of the soil. (Fig. 4, B.) Sclerotial formation was abundant under the leaf sheaths. The organism was reisolated from the sclerotia and diseased tissue.

Pathogenicity tests have also been successful when performed in the field. Sclerotia from several different cultures were placed outdoors in the soil early in the summer of 1931. Kharkof and Turkey Red wheat was planted early in the fall where the sclerotia had been all summer. The following spring, 1932, from 50 to 100 per cent of the wheat was killed

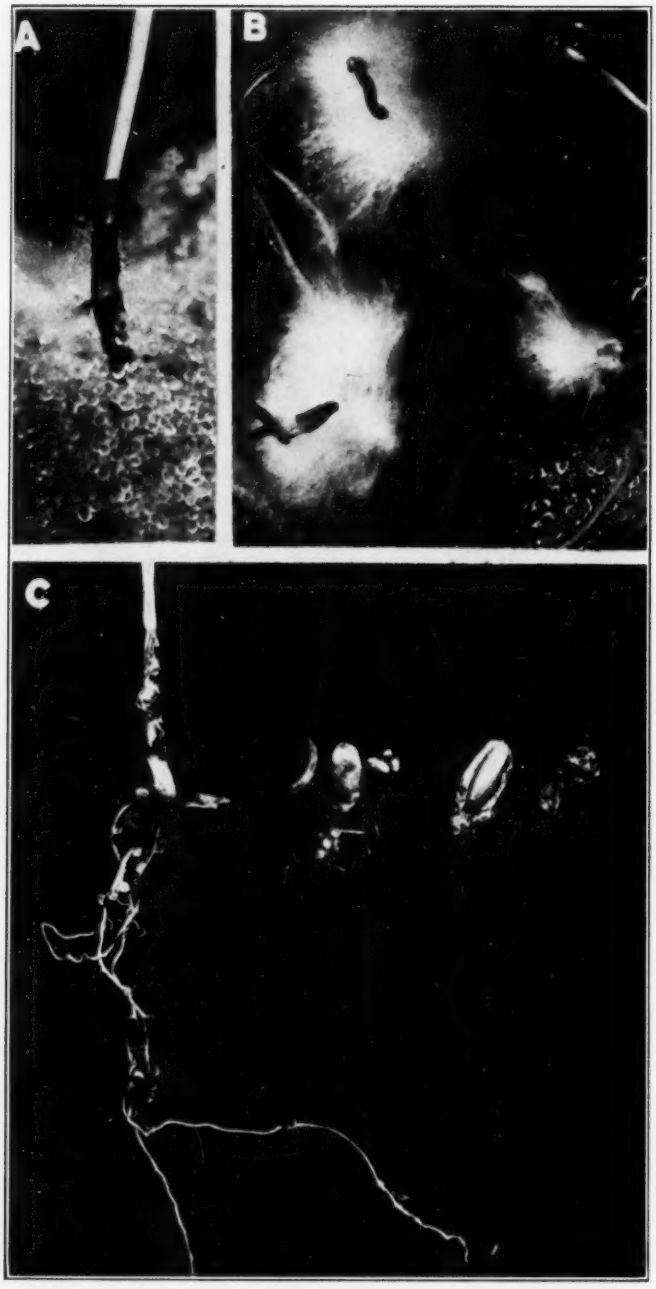

FIG. 4. A. Wheat plant inoculated with culture No. 282 growing in sterilized Ottawa silica sand. Note sclerotia embedded in the leaf sheath of the plant. B. Three wheat plants growing in soil containing sclerotia and secured from badly diseased wheat plots at Sandpoint, Idaho. Wheat was planted in this soil after it was stored out of doors during the summer. Note the mycelia on the roots. B. On the left is wheat plant shown in A after removal from the sand. Sclerotia are embedded in the leaf sheath and attached to the roots. The three seedlings on the right were killed at germination.

where planted over some of the cultures. (Fig. 2.) Culture 226 caused partial killing of plants, while cultures 277, 278, 279 killed all plants outright. The symptoms produced were identical with those found originally in the fields. The other cultures produced no symptoms.

Superintendent J. H. Christ of the University of Idaho Substation Farm at Sandpoint conducted an experiment in 1932 designed to test the susceptibility to the snow-scald fungus of the various winter barleys grown on the Substation Farm. After the various barley varieties were planted, soil was gathered from areas where the disease had been especially severe the year before and was scattered broadcast over the barley plots. A snow fence was constructed in order to keep the snow on the inoculated areas. As a result of the inoculation, the barley plots where the soil was applied were entirely killed, while the noninoculated plots, although very severely damaged, were not entirely destroyed. Evidence so far would indicate that there is considerable difference in susceptibility among the various winter wheats grown on the Sandpoint Substation Farm. As indicated above, however, none of the barleys grown possessed sufficient resistance to withstand the heavy inoculation given them, although the noninoculated seedlings showed a varietal resistance.

CONTROL

Würtenberger (16) found that applying sodium nitrate, leuna saltpetre, or sulphate of ammonia to the surface of the soil followed by thorough harrowing would keep the fungus under control. Bokura (1) recommended spraying with Bordeaux mixture just before snowfall. He found that other fungicides, such as copper-soap solution and lime-sulphur mixture, were also effective. He observed that grain sowed either early or late was particularly badly infected, but that resistant varieties could be safely sown at any time. Crop rotation is suggested by Müller (6).

Experience has shown that in the high-altitude regions of Teton Basin, Idaho, early-seeded wheat is more severely injured than that sown later in the season. A fair stand may be secured even in epiphytotic years when wheat is seeded fairly late.

SUMMARY

Diseases of grain and grasses produced by several sclerotial fungi that appear in the field after the snow melts have been increasing in occurrence and importance in the high-altitude regions in Idaho during the last few years. Fourteen cultures have been obtained of these organisms whose optimum temperatures for growth range from 0° to 10° C. Although attempts have been made to obtain the perfect stage, these have not been successful. These fourteen fungi have been placed in four groups according to their morphological and physiological characteristics. One of these

groups corresponds in growth characteristics with a culture of *Typhula graminum*, supplied by H. Tasugi, of Japan. Pathogenicity of several of these organisms has been established both by laboratory and field inoculations. The disease is best controlled by late seeding and also by using resistant varieties of cereals.

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BLACK ROT OF COFFEE IN MYSORE

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Coffee diseases, similar to the "black rot," or "koleroga" as it is called in Mysore, have been known to occur in various tropical countries, such as Venezuela, Jamaica, New Caledonia, the Belgian Congo, Puerto Rico, and Guatemala. The symptoms of the disease occurring in Mysore, the conditions under which the causal fungus thrives, and a description of the fungus have been given by Coleman, Venkata Rau, and Narasimhan (6) and by Butler (5).

The fungus grows on the dorsal surface of the leaves and over the berries in the form of a web or pellicle, which is shiny gray when dry and can be easily peeled off when wet. On the petioles, and to some extent on the twigs, it can be traced, extending as a thin ribbon. The affected leaves and berries turn brownish at first and, later, black. The blackened leaves hang

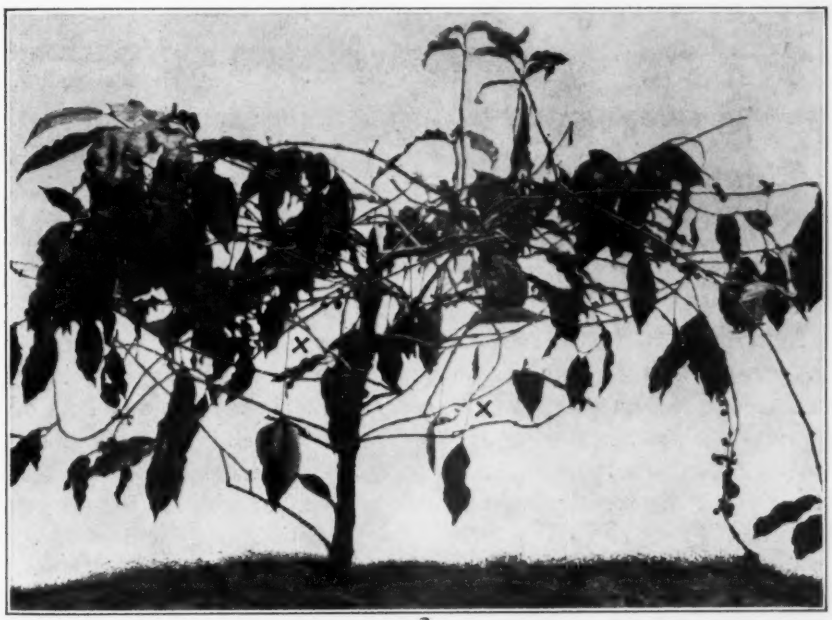

FIG. 1. Coffee bush attacked by *Corticium koleroga* showing some of the leaves suspended by mycelial strands at X.

[1] The writer wishes to acknowledge his indebtedness to Dr. Leslie C. Coleman, Director of Agriculture in Mysore, for the helpful suggestions given in the course of the investigation, which was carried on at the Coffee Experimental Station, Balehonnur.

suspended on the tree by means of the fungus strands connecting the petiole and the stem (Fig. 1). A brownish juice oozes from the leaves after decomposition. In attempting to determine the cause of this blackening, interesting observations on the life history of this fungus were made and are here presented.

Generally considered, it has to be admitted that investigations on this type of disease, which has been reported on coffee in various parts of the world, have not been complete, with the result that varying opinions have been expressed by investigators as to the identity of the fungus and the course of the disease.

Cooke (7) first named it *Pellicularia koleroga* and illustrated a type of spore that Fawcett (9) proved to belong to another fungus associated with the disease; Ernst (8) examining the Venezuelan specimen, named the fungus *Erysiphe scandens,* owing to its superficial nature. Von Höhnel (11), though he had only herbarium specimens available for examination, gave an accurate description of the basidia and basidiospores formed by the fungus and named it *Corticium koleroga.* These basidiospores, which are easily dislodged from the sterigmata, were first illustrated by photomicrographs by Coleman *et al.* (6).

MATERIAL AND METHODS

Affected coffee leaves were collected during the heavy monsoon, showing different stages of the disease, viz., leaves with grayish white film, those showing partial browning, and those that have become entirely black. The materials were fixed in Bouin's fixing solution. At first a difficulty was encountered, viz., that the fungus film soon separated from the leaf during the process of washing. This was obviated by dipping the material in melted ½ per cent agar before placing in the fixing fluid. For studying the fungus film itself, the following method was adopted:

A piece of affected coffee leaf was placed in water till the film, aided by careful manipulation with a needle, separated from the leaf and floated on the surface. It was drawn onto a slide, fixed on the slide itself for 24 hours in Bouin's fixing solution, and carefully washed in repeated changes of water. After the excess water was removed, 0.2 per cent celloidin in equal quantities of ether and alcohol was poured over it and allowed to dry. It was found that the film could, after this treatment, be safely stained in Heidenhain's iron-alum haematoxylin, dehydrated, and mounted in balsam.

MORPHOLOGY OF THE FUNGUS

The course of the disease can be divided into 2 distinct stages, viz., one in which a uniform white film is seen covering the dorsal surface of the leaf (Fig. 2, A), the other, in which hyphal clumps are found scattered all

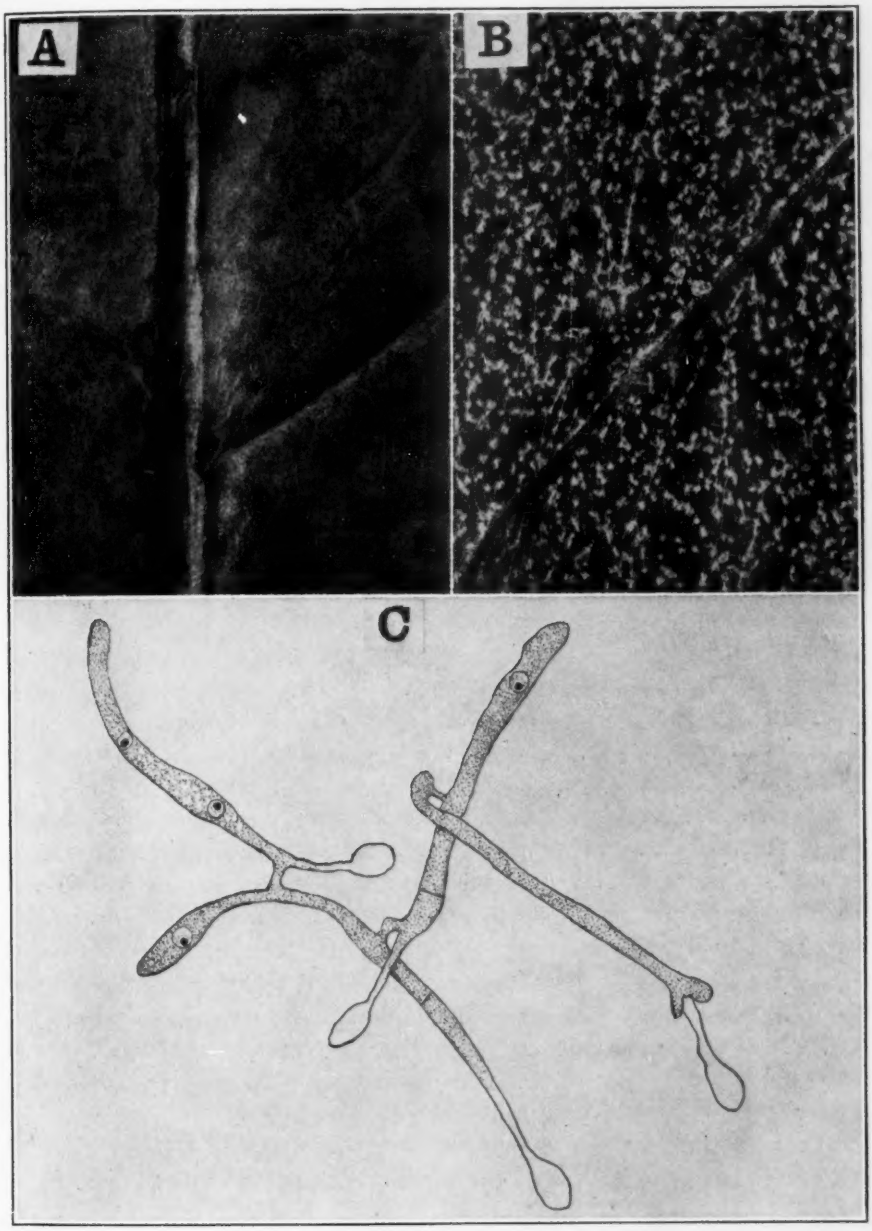

FIG. 2. Mycelium of *Corticium koleroga*. A and B. On diseased coffee leaves. A. Note the uniform whitish film on the dorsal surface. ×4. B. Note the hyphal clumps or sclerotia scattered with thin intervening spaces all over the lower surface. ×6. Photographed by Dr. Leslie C. Coleman. C. Anastomosis of hyphae from different spores.

over the affected leaf, the intervening mycelium being thin (Fig. 2, B). These may be differentiated as the early pellicle stage and the late sclerotial stage.

The Pellicle Stage. During the early stages of the disease, the film, when wet, can be peeled off the leaf, but it does not come off easily on areas where the leaf has already turned brown. It is made up of a mass of interwoven threads, "with here and there a primary strand consisting of a number of hyphae running parallel to each other" (6). The color of the mycelium is hyaline. No clamp connections or anchor cells, as in the case of "thread blights," have been found. Instead of these, repeated hyphal fusions have been found to be formed between the threads. One of 2 hyphae lying close together develops a lateral branch, which fuses with the other hypha and establishes a connection between them; or, where 2 hyphae cross, fusion by means of a short connecting branch occurs close to the angle formed by the 2 hyphae. Such connecting branches of 2 different hyphae have been found in *Panus stypticus* and *Coprinus lagopus* by Buller (3, p. 154) and named by him "bridging hyphae," and in *Hypochnus solani* by Müller (13).

The basidia are formed at the ends of lateral branches, being round or ovoid and measuring 8.5–12 μ in diameter, and are not found in clusters of 3 or 4, as Von Höhnel (11) reported. The basidiospores, borne on long tapering sterigmata, have been described and photographed by Coleman *et al.* (6). Fully developed spores from fresh material measured 9.1 μ × 3.4 μ. In no instance were they found to measure 13 μ in length, as reported by Burt (4). The length of the sterigmata was reported to be 2 μ by Von Höhnel (11) and 9–11 μ as measured on fresh material by Coleman *et al.* (6). It was found that the length of the sterigmata was inconstant, varying from 5 μ to 11.5 μ. Figure 3, B, shows short sterigmata bearing fully developed spores, while in figure 3, A, it is seen that the sterigmata bearing immature spores are long and tapering and of unequal lengths. The spores are easily dislodged and lie about in the meshes of the film. They are slightly flattened on one side, rounded at one end, and somewhat pointed at the other. Abundant spore formation has been noted in the estate only at the time when the humidity is high. The best method of studying the spores is the one followed by Coleman *et al.* (6), viz., attaching pieces of affected leaves bearing basidia to the inner surface of the Petri-dish cover and allowing the spores to drop on agar contained in the bottom dish. In heavy monsoon weather, the spores have been found to drop in 4 hours' time and germinate 2 hours later, forming germ tubes with swollen ends (Fig. 3, C).

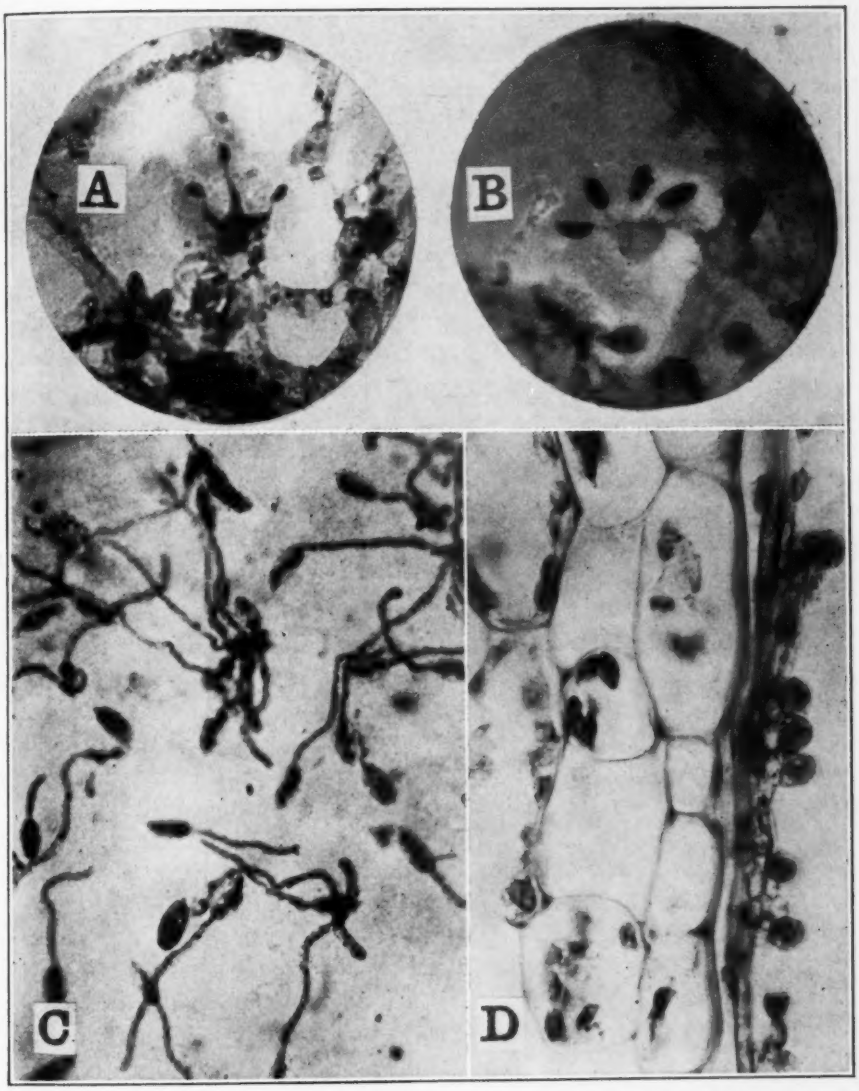

FIG. 3. A and B. Photomicrographs of basidia of *Corticium koleroga*. ×900. Note the immature basidiospores and tapering sterigmata of unequal lengths in A and the fully developed basidiospores and short and equal-lengthed sterigmata in B. C. Germinating basidiospores. ×1,350. D. Section of coffee leaf with fungus film on its lower surface. ×900. Note the nuclei in some of the basidia.

When a large number of spores germinate side by side on agar, anastomosis has been observed often between the hyphae formed by the germination of 2 different spores. Sometimes, adjacent threads developing from

2 different spores fuse, at others the threads from 2 spores lying far apart. This has not been observed to take place during the early stages of germination but only after the germ tubes have grown to some extent. Both terminal and lateral fusions have been observed (Fig. 2, C). Müller (13) reported similar hyphal fusions of germinating spores in *Hypochnus solani*. Buller (3) reported the occurrence of such hyphal fusions of germinating spores not only in the Hymenomycetes but in some of the Ascomycetes, viz., *Trichoderma lignorum, Pleurage anserina,* and *Ascobolus magnificus,* and in one of the Fungi Imperfecti, viz., *Colletotrichum trichellum*. The significance of these hyphal fusions, as Buller pointed out, lies in the fact that the conduction of food materials to the sclerotia and fruit bodies is facilitated and that the association of nuclei in conjugate pairs is thus made possible.

The spores as well as the hyphae resulting from germination are uninucleate. After fusion, the hyphae, which develop further, are always binucleate. Similar fusions leading to the binucleate stage have been reported by Willy Harnack (10) in the case of *Schizophyllum commune* Fr. and *Collybia tuberosa* Bull.

Isolation of the Fungus. Spores were made to drop on onion agar, following the method of Coleman *et al.* (6). These subsequently developed into a hyphal mat on the surface of the agar, which was transferred to tubes of various media. On oat agar and onion agar whitish aerial mycelium is produced, but on peptone sugar agar and peptone starch agar only a surface growth develops. A fairly good growth is obtained on bits of filter paper moistened with peptone starch solution recommended by Müller (13) for *Hypochnus solani*. The development of basidia has never been noted on this medium, as claimed by Müller for *H. solani*. No fructifications have ever been noted to develop on any of the media tried hitherto.

Following Bavendamm's (2) cultural experiments on wood-rotting fungi, the effect of adding to the media varying quantities of tannic acid, gallic acid, pyrogallol, and guajacol, on the growth of *Corticium koleroga* was noted. It was found that on onion agar containing 0.1 per cent tannic acid or gallic acid a brown halo was formed in advance of the growing mycelium (Fig. 4, A). On media containing 0.05 per cent guajacol, a dark reddish brown halo (Ridgway's Pompeian Red) was formed (Fig. 4, B).

The formation of a brown halo on media containing tannic acid was found by Bavendamm to be characteristic of lignin-destroying organisms, such as *Stereum purpureum* and *Trametes radiciperda*. He found that those involved in cellulose disintegration, such as *Merulius lacrymans* and *Coniophora cerebella*, did not form a brown halo. The brown discoloration, according to Bavendamm, is caused by the action of the oxidase produced

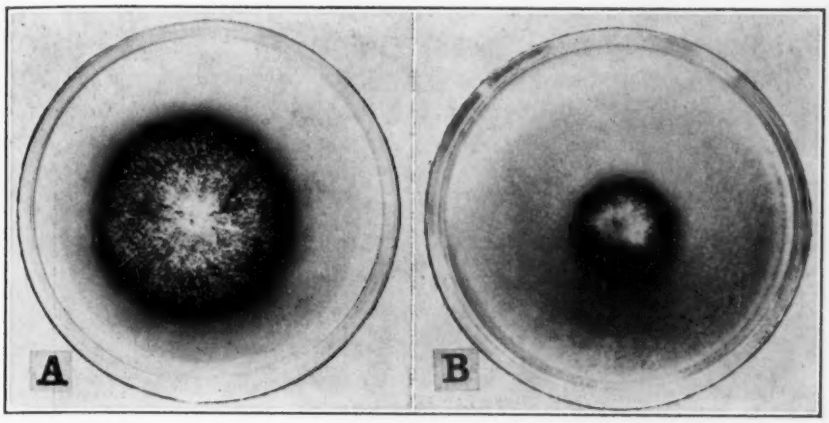

FIG. 4. Pure 6-day-old cultures of *Corticium koleroga*. A. On onion agar to which 0.1 per cent tannic acid had been added. Note the dark halo around the colony. B. On onion agar to which 0.05 per cent guajacol had been added. The dark-red halo around the colony appears black in the photograph.

TABLE 1.—*The cultural behavior of Corticium koleroga on onion agar to which various chemicals were added*

Chemical	Percentage of chemical added to the media			
	0.05%	0.1%	0.25%	0.5%
Tannic acid	Very good growth Brown halo	Very good growth Brown halo	Fair Brown halo	Poor Brown halo
Gallic acid	Good growth Brown halo	Poor Brown halo	Nil	Nil
Pyrogallol	Poor growth Brown halo	Nil	"	"
Guajacol	Fair Reddish brown halo	"	"	"

by the fungus on tannin. In cultures of *Corticium koleroga* there are definite indications of the presence of oxidase.

The Sclerotial Stage. The fluffy, pulverulent appearance of the film in course of development changes into one that is dotted with a large number of hyphal clumps, the intervening spaces being thin (Fig. 2, B). At this stage the mycelium gradually changes from a whitish to fuscous color and the affected leaf turns to a rusty brown. These sclerotia are formed by

repeated branchings of short cells and are ultimately pseudoparenchymatous in nature, the component parts consisting of short binucleate cells (Fig. 5, A). The sclerotia are hyaline during the early stages but change to a fuscous brown later. Neither Von Höhnel (11) nor Burt (4) found these structures in specimens of *Corticium koleroga* examined by them. Burt

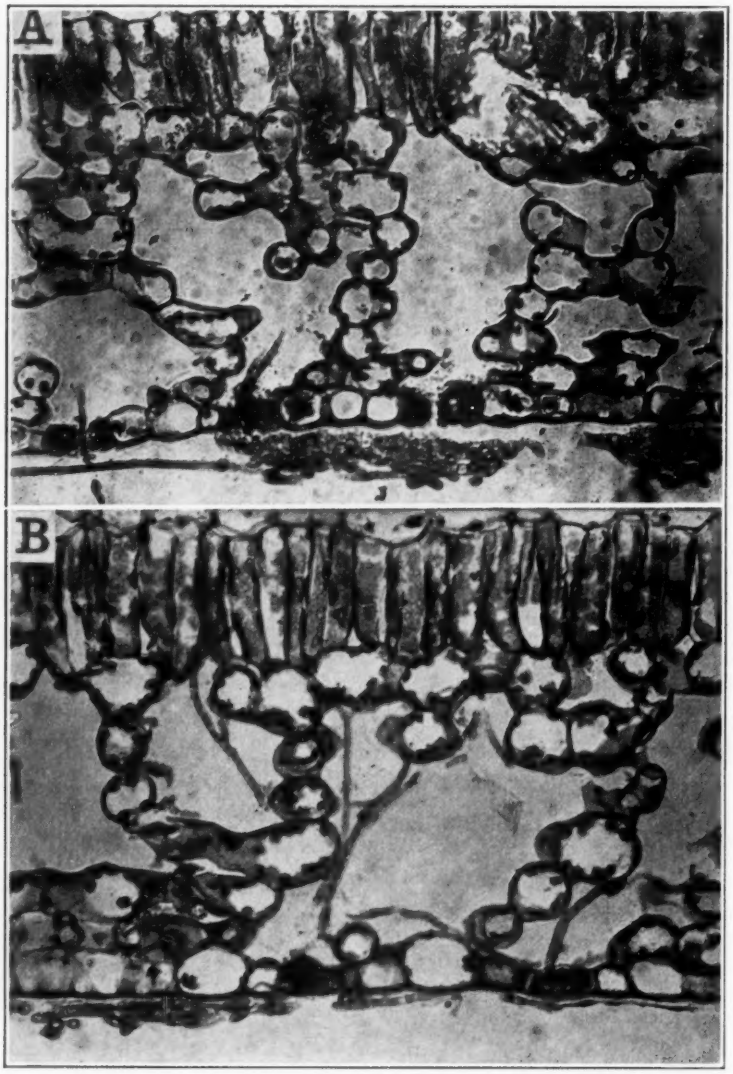

FIG. 5. Sections of coffee leaves infected with *Corticium koleroga*. ×300. In A, note the sclerotia and the penetration of fungus through the stomata. In B, note the intercellular mycelium.

(4), however, noted their presence in *C. Stevensii*, parasitic on leaves and branches of apple, pear, and quince. Wolf and Bach (14) found these structures sparsely on grapefruits affected by thread blight and in abundance on affected pears, apples, pecans, and pomegranates. These were first reported in *C. koleroga* on coffee by Coleman *et al.* (6). The sclerotia carry the fungus through the dry season.

From descriptions given in literature of similar coffee diseases in the tropics, one is tempted to conclude that these structures are characteristic of the disease. Kuyper (12), describing the "*Zilverdraadziekte*" of coffee in Surinam, reported on the occurrence of "Hyphen Kluwens" peculiar to this disease. Bally (1) compares these with the hyphal-clump stage ("*Knobbeltjesvorm*") in the disease known as *Djamoer Oepas* on coffee in Java. In his comparative study of the coffee diseases occurring in Mysore, Puerto Rico, and Venezuela, Fawcett (9), who did not recognize the basidiomycetous nature of the fungus, described and illustrated structures that he called holdfast cells. Burt (4) gave his opinion that these were probably young basidia. The writer is inclined to believe, judging from the figures, that they are early stages of sclerotia. The small hold-fast cells, which, according to Fawcett, differentiate the candellilo disease of coffee in Venezuela from other diseases of similar nature are comparable to the tiny clumps of sclerotia found during the early stages in Mysore.

PENETRATION OF THE FUNGUS INTO THE COFFEE LEAF

The question whether this type of fungus penetrates the tissues has not been satisfactorily investigated. Von Höhnel (11), who examined the specimens at Kew, reported that the fungus was entirely superficial. Burt (4) who based his comprehensive description of *Corticium koleroga* on a small portion of the Venezuelan specimen and microscopic preparations of the Mysore specimens sent to him from Kew, reported the presence of intercellular vegetative mycelium. He could not have examined affected tissues, as, from his own words, only preparations of the basidiospores and hyphae were sent to him from Kew. Coleman *et al.* (6) found no evidence of penetration. They suggested that the death of the leaves, as evidenced by the blackening, might be due partly to reduced respiratory activity owing to blocking of the stomata and partly to the secretion of an enzyme or toxin. Butler (5) thought it probable that there was a direct absorption of nutrition by the parasite by osmosis from the leaf. Thus, it will be seen that varying opinions have been held as to whether or not the fungus penetrates the tissues. In order to settle this question the writer examined a large number of hand and microtome sections of affected leaves in various stages of the disease.

Leaves covered on the dorsal surface with the uniform whitish film, *i.e.*, in the early stage when basidia were formed, have never shown the presence of intercellular mycelium in the tissues (Fig. 8). No trace of any sort of injury to the epidermis can be noted. Any evidence of penetration into the tissues can be made out only during the later stage, when hyphal clumps are formed. Sections of leaf areas that have turned rusty brown, show, attached to the lower epidermis, compact masses of pseudoparenchymatous cells (Fig. 5, A).

The hyphae emerging from these masses penetrate the tissue through the stomata (Fig. 5) and permeate the spongy parenchyma, often reaching as far as the palisade cells. In leaf areas that are adjacent to a brown patch and still green, the formation of the sclerotia is to be seen, with occasional penetration of the tissues. The brown color appears in the leaf only after penetration of the tissues by the hyphae. The secretion of oxidase by the fungus that penetrates the tissues of the leaf perhaps causes the blackening of the leaf. During later stages of the disease, the leaves begin to decompose because of invasion by bacteria, and a brownish liquid oozes out.

It is often seen that during very heavy rains some of the leaves turn black, though no fungus is noticeable on them. This is due to the fact that the leaves get water-soaked and become decomposed by bacteria.

COMPARISON WITH SIMILAR DISEASES OCCURRING IN OTHER PARTS OF THE WORLD

Our imperfect knowledge of the complete life history of this type of disease on coffee, occurring in various parts of the tropics, led investigators to believe that some of these are different from the type occurring in Mysore. Fawcett (9) found that the Mysore and Puerto Rican diseases were similar but different from the Venezuelan disease.

Reviewing our present state of knowledge of these diseases, Bally (1) says that the question "Whether the spinnewebeziekte of Java, the Zilverdraadziekte of Surinam, the candellilo of Venezuela, the koleroga of India, and perhaps a disease which Bernard in 1907 described on tea, are caused by the same fungus, remains to be settled." Of these, the Java disease is said to be different, owing to the presence of anchor cells, and is classified under the Marasmioideae group; but, as Bally pointed out, the disease on tea described by Bernard and *Corticium koleroga* on coffee are almost identical, except for the light pink color of the former.

He however expressed the opinion that the *zilverdraadziekte* of South America is not identical with any of the described kinds found in the East, as it differs from *Corticium koleroga* in that its mycelium enters by way of the stomata and develops hyphal clumps.

In view of the writer's finding in the Mysore disease that the sclerotia-forming mycelium penetrates the leaf tissues, there seems to be no ground

for considering the South American disease different. Very little is known of the life history of the candellilo disease of Venezuela. Fawcett (9) considered it to be different from the Mysore and Puerto Rican diseases, because of the smaller holdfasts. From an examination of the figures given by him, it may be concluded that these holdfasts are probably early stages of sclerotial formation. When more is known about this disease, it will probably be found that it is identical with the Mysore disease.

The facts presented above are, in some important respects, at variance with those presented by Burt (4) in his description of *Corticium koleroga*. He pictured the course of the disease as follows:

"The parasitic vegetative mycelium forms long slender mycelial strands of rather uniform diameter, whitish or pallid at first, finally fuscous, running along the branches and midrib and veins of the leaves, infecting the leaves, and ramifying between the cells of the leaf-parenchyma, *finally*[2] emerging at many points on the under side of the leaf to form minute fructifications, soon laterally confluent into a thin arachnoid, perforate membrane covering the under surface of the leaf between the midrib and the principal veins."

The above description leads one to believe that the penetration of the tissues by the fungus takes place prior to the formation of the basidia and the basidiospores. Penetration by the hyphae that bear basidia has never been observed by any of the investigators. As seen in figure 3, D, the fungus at this stage is entirely superficial. It is only later, when it forms the sclerotia, that penetration is in evidence. Basidia have never been observed to form after the fungus penetrates the tissues. As to what the fungus feeds on during the pellicle stage it is difficult to say unless it is assumed that there is absorption of nutrition by osmosis.

Control Measures. The disease has been satisfactorily controlled by spraying with Bordeaux mixture (5:5:50) before the outbreak of the monsoon. The use of an adhesive, such as lime caseinate, has been found to be essential in order to prevent the mixture from being washed off by the heavy rains. An area of 12,000 acres of coffee was sprayed during the year in Mysore.

SUMMARY

Corticium koleroga (Cooke) Von Höhn. causing black rot of coffee in Mysore, has two distinct developmental stages, viz., the early pellicle stage in which a white uniform film is seen covering the under side of the leaf and the late sclerotial stage in which sclerotia are found scattered all over the dorsal surface.

[2] Italicized by the writer.

Basidia and basidiospores are to be observed only during the pellicle stage of development. The fungus is entirely superficial at this stage. Bridging hyphae are commonly seen.

Anastomoses of the hyphae formed by the germination of the basidiospores have been observed.

By adopting Bavendamm's cultural method it has been found that *C. koleroga* produces oxidase during growth.

Penetration of the leaf through the stomata by the fungus has been observed only during the late sclerotial stage. The blackening of the leaf is considered due to the secretion of oxidase into the tissue by the fungus.

A comparison between the black rot of coffee in Mysore and similar diseases on coffee occurring in other parts of the world shows that there is not sufficient ground for considering any of them different except the thread blight occurring in Java.

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A MOSAIC OF THE FIG IN CALIFORNIA[1]

IRA J. CONDIT AND W. T. HORNE

(Accepted for publication Dec. 27, 1932)

The foliage of fig trees in California is, in general, free from serious diseases such as leaf rust, *Kuehneola fici* (Arth.) Butler, which is an important factor in the commercial production of figs in many other districts. There has long been recognized here, however, a mosaic-like trouble of fig leaves, which occurs on practically all varieties wherever grown in this State. It appears to have been almost totally overlooked in the pathological literature of the fig both in this and in foreign countries. This leaf trouble is sufficiently severe in some cases to cause not only leaf drop but fruit drop, as well, and, therefore, warrants consideration from various standpoints such as variety resistance or immunity, possible insect vectors, and breeding of resistant types.

Fig diseases have been discussed by various writers ever since the time of Theophrastus (7, Vol. I, p. 401) third century B. C., who mentions a "scorching" of branches and foliage due to cold winds. Pellicano (3) describes a leaf scald of fig trees in Italy in which leaves turn yellow and dry up prematurely on account of drought, as well as chlorosis of foliage due to obscure causes. Vallese (8) in his book "Il Fico" discusses a chlorosis or yellowing of fig leaves and suggests that sprinkling with a weak solution of iron sulphate may produce beneficial results.

A leaf scald of Sicilian fig trees is mentioned by Portale (4) as being similar to the "squalor malorum" of Theophrastus. A light yellowing of the leaves is first observed, which is soon intensified even in the fruit, ending later with the shrivelling and dropping of both leaves and fruit. It appears to be due, according to Portale, to prolonged summer heat. L. Savastano (5) gives a brief account of a chlorosis of fig leaves in August following drought, the chlorosis being diffused over the leaf surface and not localized in spots.

None of these references to fig culture in Italy, however, describe any leaf troubles that produce mosaic-like spotting of the foliage.

A single reference to mosaic of fig leaves has been found in American literature, that of Swingle (6), who states:

"Many years ago I secured at Biskra, in the great date oasis of the Algerian Sahara, cuttings of a fig which has proven to be very well adapted to hot, dry, irrigated valleys in the Southwest. It is, so far as I know, the

[1] Paper No. 291, University of California Graduate School of Tropical Agriculture and Citrus Experiment Station, Riverside, California.

only fig among twenty-five or more varieties tested in the hot irrigated valleys where dates can be grown successfully, which does not show more or less severe mottling. It seems to be quite unaffected by the intensely hot weather in midsummer, the leaves remaining vivid green instead of being yellowed by mosaic mottling."

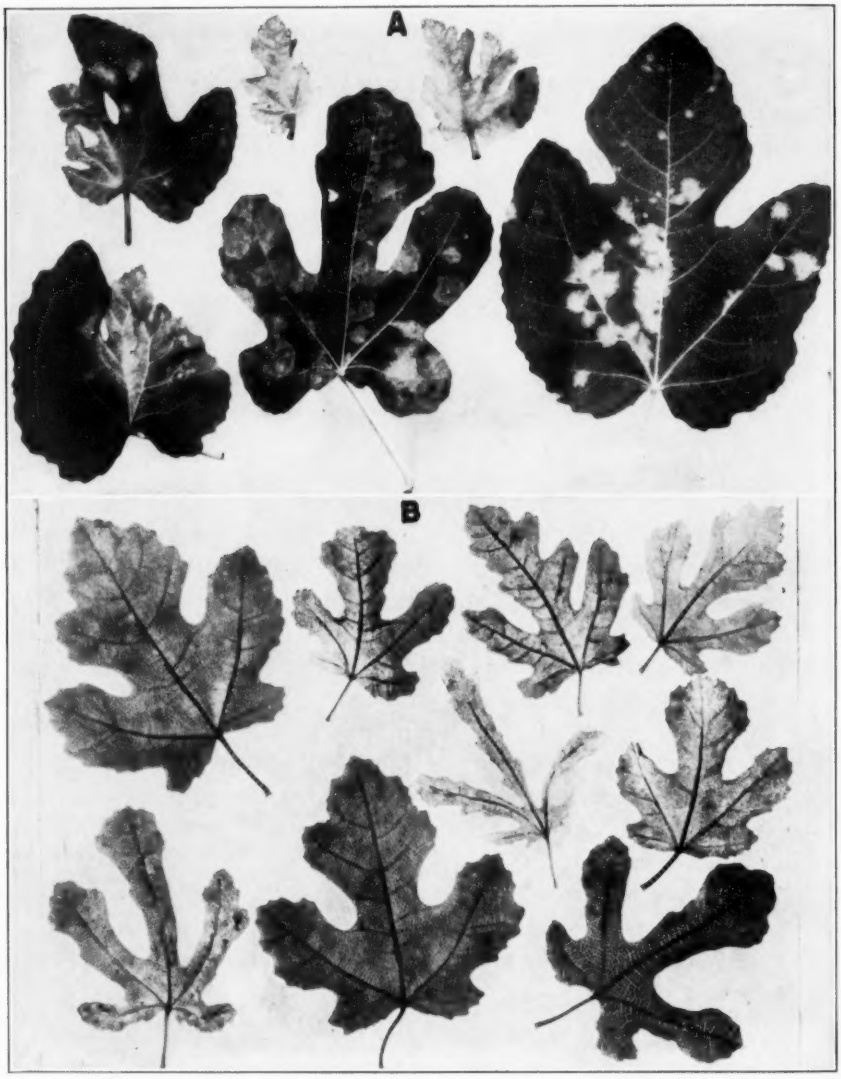

FIG. 1. A. Fig mosaic manifests itself not only as distinct yellowish spots on the normally green leaves but also in various malformations of the leaves. B. Budlings from healthy seedlings, growing on mosaic-infected trees, produce leaves with typical mosaic spots and malformations.

Hodgson has recently (2) reported that a mosaic of fig leaves is widespread in Tunisia but is of no economic importance.

MOSAIC SYMPTOMS

Fig mosaic manifests itself both on the leaves and on the fruit. On the leaves the mosaic spots are sometimes sharply delineated, the light yellow color of the affected areas contrasting strongly with the normal green color of the foliage (Fig. 1, A). The borders of mosaic spots are, however, usually indefinite, their light color blending gradually with the dark green of healthy tissue (Fig. 2, A). In some leaves the spots are of more or less uniform size, small, and densely scattered over the surface. In other cases they are of various sizes, of indefinite outline and appearing as irregular patches of light green, diffused widely throughout the blade of the leaf with no relation whatever to the position of the leaf veins. Mosaic spots often are bordered by a rust-colored band, evidently caused by the death of epidermal or subepidermal cells. Seldom does the mosaic bring about an actual necrosis or dying out of whole areas of leaf tissue.

One manifestation of fig mosaic, which appears to be systemic, is the malformation of leaves. In some cases certain twigs bear none but malformed leaves. In other cases both malformed and normal leaves are found on the same twig. Malformed leaves show an infinite variety of shapes and sizes (Fig. 3, B).

The appearance of mosaic spots on the fruit is very similar to that on the leaves (Fig. 2, B). The premature dropping of figs appears at times to be due to the presence of these mosaic spots on the surface of the fruit and on adjacent leaves.

VARIETY SUSCEPTIBILITY

In the commercial fig districts of California mosaic apparently is not sufficiently serious to alarm the growers. Of the 5 leading commercial varieties the Kadota and Calimyrna are least affected by mosaic, the deep green foliage showing little if any mottling. The White Adriatic and the Brown Turkey, under good cultural conditions, are apparently little affected, although the presence of deformed leaves due to mosaic is common on trees of the former variety. The foliage of Mission trees is quite commonly seriously mottled by mosaic, often sufficiently so to interfere with photosynthesis and later to cause leaf drop; its presence on the figs may result in improper coloring of the fruit in drying.

Among the hundred or more varieties of figs in a variety planting at the University of California Citrus Experiment Station, Riverside, there are striking variations in the degree of susceptibility to mosaic. For convenience the varieties can be classified into 4 groups, depending upon the susceptibility

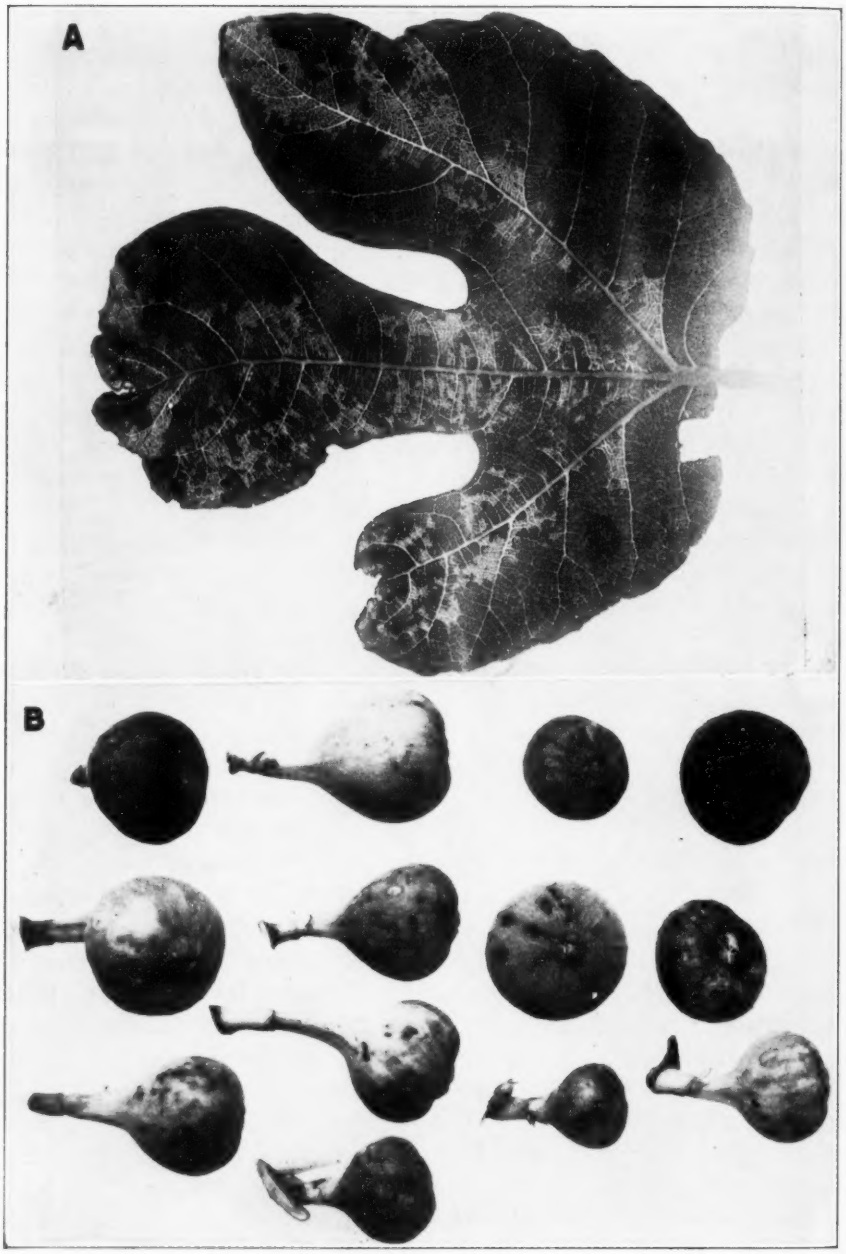

FIG. 2. A. In this fig leaf the mosaic patches are rather indefinite and not sharply delineated. B. Fig fruits, as well as leaves, show mosaic spots.

of leaves and fruit to this trouble; those immune or essentially so, those very slightly affected, those slightly affected, those appreciably affected.

There is only one variety that can be called almost immune from mosaic—an entire-leaf caprifig form of *Ficus palmata* Forsok. A careful

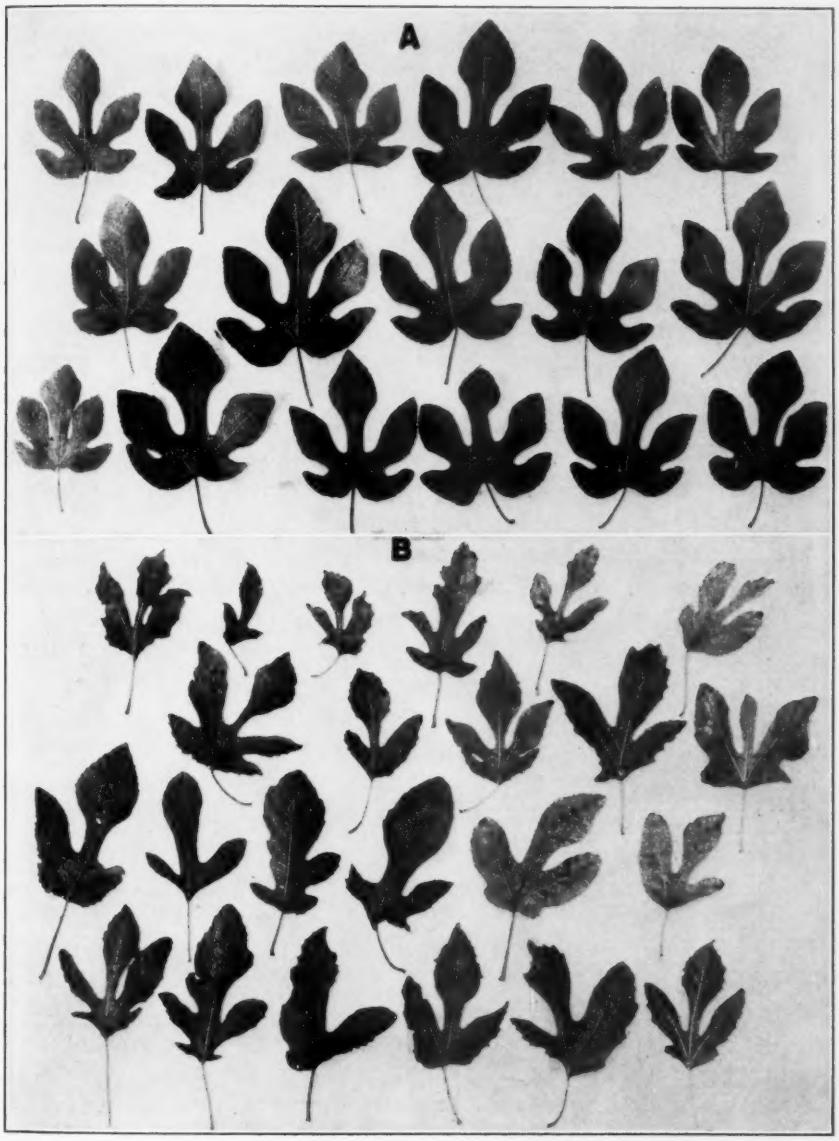

FIG. 3. Leaves from same Adriatic tree. A. Normal. B. Malformations due to mosaic.

search of 2 vigorous trees failed to show any leaves certainly affected by mosaic, although there were indications of its presence on a few leaves. Some seedling fig trees having *F. palmata* as the male parent appear to show immunity from mosaic.

In group 2 there are at least 4 varieties, very slightly affected by mosaic. The Biskra is a very vigorous tree, with the largest leaves of any variety studied, but occasional leaves show unmistakable mosaic spots.

The Hamma, which appears to be a form of *Ficus palmata* bearing edible fruit, is very slightly affected, but shows some indistinct mosaic areas in the leaves. A lobed-leaf caprifig form of *F. palmata* is affected about the same as the Hamma. Of all the varieties of common figs in the plot the Pastiliere is the least affected, only scattered spots appearing on fruit or leaves.

The Calimyrna, Kadota, Kassaba, White San Pedro, and Roeding No. 1 caprifig are varieties showing very apparent mosaic spots, but the color of the foliage is not sufficiently affected to materially reduce photosynthetic activity. At least, there is no apparent lack of tree vigor traceable to the presence of leaf mosaic. The Brunswick (Magnolia) foliage is slightly affected. Some cases are found where the immature Brunswick fruit itself is affected sufficiently to account for its premature abscission. In these cases the variety would logically fall into the next group.

The number of varieties very appreciably affected by leaf mosaic is large. No attempt is made here to list or describe all those represented in the collection but simply to point out some that appear especially susceptible. Considerable malformation of leaves is present in one caprifig tree of *Ficus pseudocarica* Miq., the malformed leaves, however, being restricted to the least vigorous branches.

Mosaic spots are very prevalent on both leaves and fruit of the White Ischia and the Celeste varieties, the latter being sufficiently affected to account for the premature shedding of fruit. The Cheker Injir, a Smyrna type fig, is very susceptible, the surface of the fruit being spotted with light green areoles, which later become rusty.

Some varieties, such as Baalie and Panache, show numerous leaves malformed by mosaic, the malformations in some cases dwarfing the entire leaf. Malformed leaves are especially common on Roeding No. 3 caprifig. De Jerusalem leaves have very pronounced and striking mosaic spots, which early develop necrosis; the immature figs are also markedly affected. In the White Adriatic the mosaic spots are widely diffused over the leaf surface and the trouble appears to be sufficiently serious to account for the dropping of terminal leaves (Fig. 4, B). Deformation of small green figs of certain varieties, such as Sultane, is attributed to the presence of mosaic.

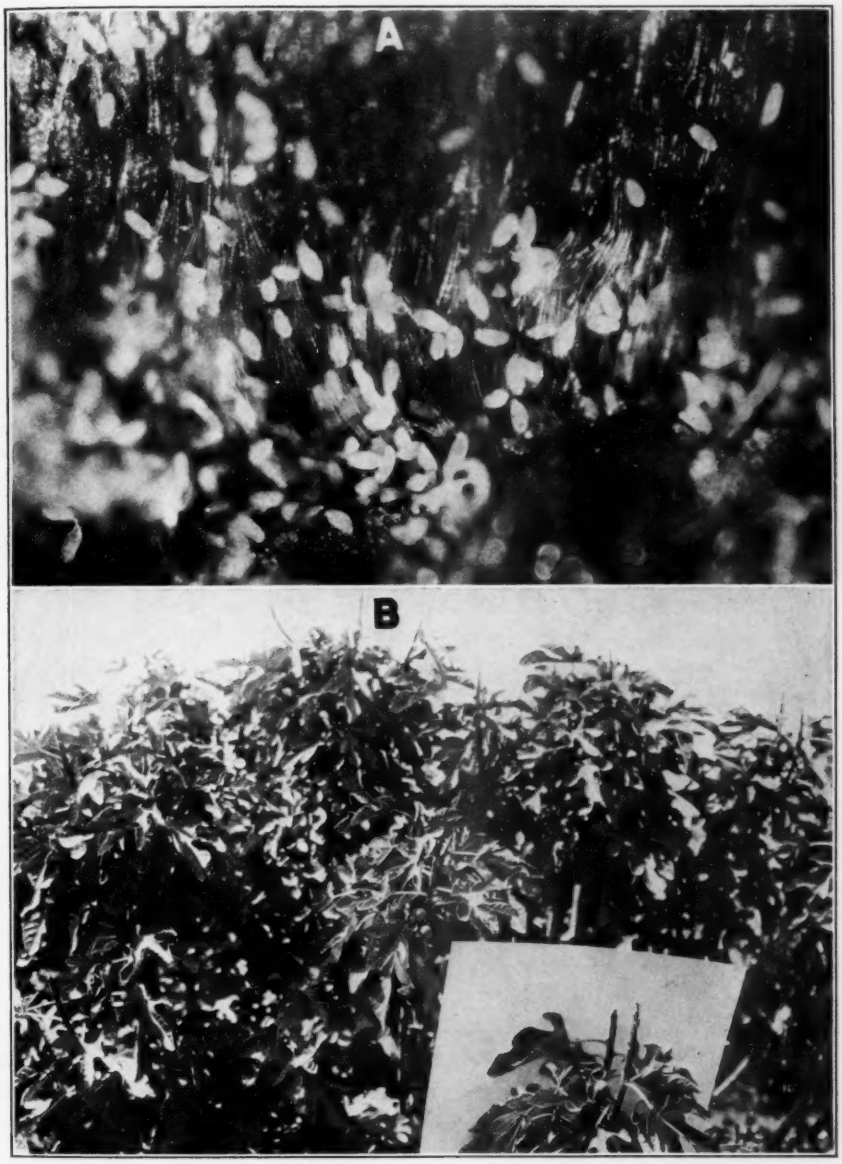

FIG. 4. A. Fig mites (*Eriophyes fici*) often infest bud scales and young leaves in immense numbers, sometimes causing the abscission of leaves and stunting of twigs. B. In some varieties, such as the Adriatic, fig mosaic appears to account for the early defoliation of terminal twigs.

POSSIBLE VECTORS

Insect pests are commonly suspected of causing malformations in leaves or of transmitting virus diseases when present. The foliage and branches of fig trees in California are not commonly infested with insect or other pests. The Mediterranean fig scale (*Lepidosaphes fici* Sign.) has become established in Fresno and Tulare Counties (California) and is rapidly spreading through commercial orchards. Certain species of thrips are at times found on the tender foliage but apparently do not injure the leaves to any appreciable extent. They are, however, often responsible for fungus infection of the fruit interior and for its ultimate spoilage (Hansen, 1). Aphid infestations of fig foliage are rare and evanescent and are seldom if ever injurious.

In 1920 an arachnid, *Eriophyes fici* Ewing, was found to be very prevalent in fruits and on terminal bud scales of figs at Fresno. This mite is now known to be present on fig trees in all parts of California and in some localities in Oregon. Infestations of terminal buds (Fig. 4, A) are often sufficiently serious to cause the drying up and dropping of very small immature leaves and the stunting of twigs. In a letter dated September 16, 1932, J. R. Watson reports that he has noticed fig mites in Florida and in one case the infestation was sufficiently severe to cause all leaves to drop from fig trees at Gainesville. In the fig districts of Mississippi, Louisiana, and Texas neither fig mites nor mosaic appears to attract attention, possibly because of heavy pruning and the resultant vigorous succulent growth. Under date of October 27, 1932, Dr. G. Grandi, Lab. di Entomologia, Bologna, writes that neither fig mites nor foliage mosaic of fig trees is mentioned in Italian literature.

Recent conferences with certain specialists fail to show any reference in literature to the transmission of a virus disease by mites. In the disease under discussion it is possible that the spots on fig leaves may result from the localized feeding of mites when the leaves were young and tender. In August, 1932, however, 3 fig trees, probably of the Mission variety, were seen near Ashland, Oregon, with mites abundant on the bud scales and very young leaves but with deep green foliage, apparently entirely free from mosaic. We have suspected fig mites of transmitting the virus of leaf mosaic, but thus far our suspicions are not confirmed or supported by experimental data.

FIELD AND LABORATORY STUDIES

The fig tree is almost universally propagated by means of cuttings from mature wood. Such cuttings would naturally reproduce any disease that

is systemic in the branches. In order to throw light on the question whether the mosaic-like lesions are local disturbances or stigmanoses in the leaves and fruit, or the results of some more general disturbance in the plant body, the following experiment was carried out: Twenty-five cuttings each were taken in March from 4 different varieties of fig trees known to be seriously infected with mosaic. These cuttings were rooted in sand in a closed box in the greenhouse and by May 5 many had leaves showing mosaic symptoms. On June 8 a census was taken of the 100 rooted cuttings with the result that 74 showed mosaic certainly present, 16 showed no positive indications of mosaic, while 10 had insufficient leaf growth to enable one to draw any conclusion. Of the 100 twigs left on the tree from which the cuttings were taken, 51 showed intense leaf mosaic on June 8, 30 showed mosaic to be certainly present, while 19 could not be located.

Seedling fig trees secured from seed of mosaic-affected parents have been grown in the greenhouse up to 12 inches in height without the appearance of any discoloration resembling mosaic on the foliage.

That mosaic is readily transmissible to healthy seedlings was strikingly shown by the following experiment: Two buds from each of 5 apparently healthy seedlings were inserted in July into branches of an orchard tree showing typical mosaic spots on the foliage. Nine budlings, alive and growing late in October, all showed unmistakable symptoms of leaf mosaic, evidently transmitted from the stock to the scion (Fig. 1, B). The plants from which the buds had been taken, kept in the greenhouse and lathhouse, put out new growth, which showed no symptoms of mosaic. Healthy seedlings, on which fresh mite-infested fig leaves were put, have, in 2 cases, at least, developed unmistakable mosaic spots. In this case the mosaic may have been transferred by contact infection but other attempts to transmit it in this way have all failed.

Studies of fig trees are being continued, but the preliminary results here reported are believed to constitute valid items of evidence tending to show that we have to do with a true mosaic disease.

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BACTERIAL SPECK OF TOMATOES

MARY K. BRYAN

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INTRODUCTION

In the course of a study of bacterial spot of tomato (*Bacterium vesicatorium*) for a comparison of early symptoms on fruit with those caused by bacterial canker (*Aplanobacter michiganense*) some isolations gave pure cultures of a green fluorescent bacterium entirely different from either of the two organisms under investigation. Inoculations on young tomato fruits, made by swabbing with water suspensions of this third organism produced abundant spotting. Reisolations from the resultant infections gave the same green fluorescent organism as was used for inoculation, and this reisolation, in turn, produced the same small dark spots when swabbed on young fruits. It seems definitely established, therefore, that there is a third bacterial spot of tomato fruits heretofore confused with some stages of bacterial spot (*Bact. vesicatorium*). The name bacterial speck is proposed for this disease and the name *Bacterium punctulans*, n. sp., for the causal organism.

GEOGRAPHICAL DISTRIBUTION

Speck was first demonstrated as a new disease, as distinguished from bacterial spot, in the spring of 1931 at Bradenton, Florida. In 1930, however, the writer was shown a field in the same locality in which the larger part of the fruit was peppered with tiny black spots and no large, typical lesions due to *Bact. vesicatorium* were observed. It was supposed, however, that this was a phase of that disease, perhaps connected with sand injury. Specimens were collected and preserved but no isolations were made. Examination of these specimens in the light of present knowledge indicates that the disease was probably speck. From S. P. Doolittle it was learned that during more than one season this type of spot was more prevalent at Bradenton and did more damage than the typical spot caused by *Bact. vesicatorium*. He also observed the disease in considerable abundance in tomato plots at Madison, Wisconsin, in 1929.

Specimens infected with typical speck were also received in the summer of 1931 from Maryland. A green fluorescent organism, identical with that from Florida speck, was isolated from these and proved pathogenic.

SYMPTOMATOLOGY

The name *speck* was selected because of the minute nature of the lesions on the fruit, which seldom attain a diameter of more than 1 mm. (Fig. 1,

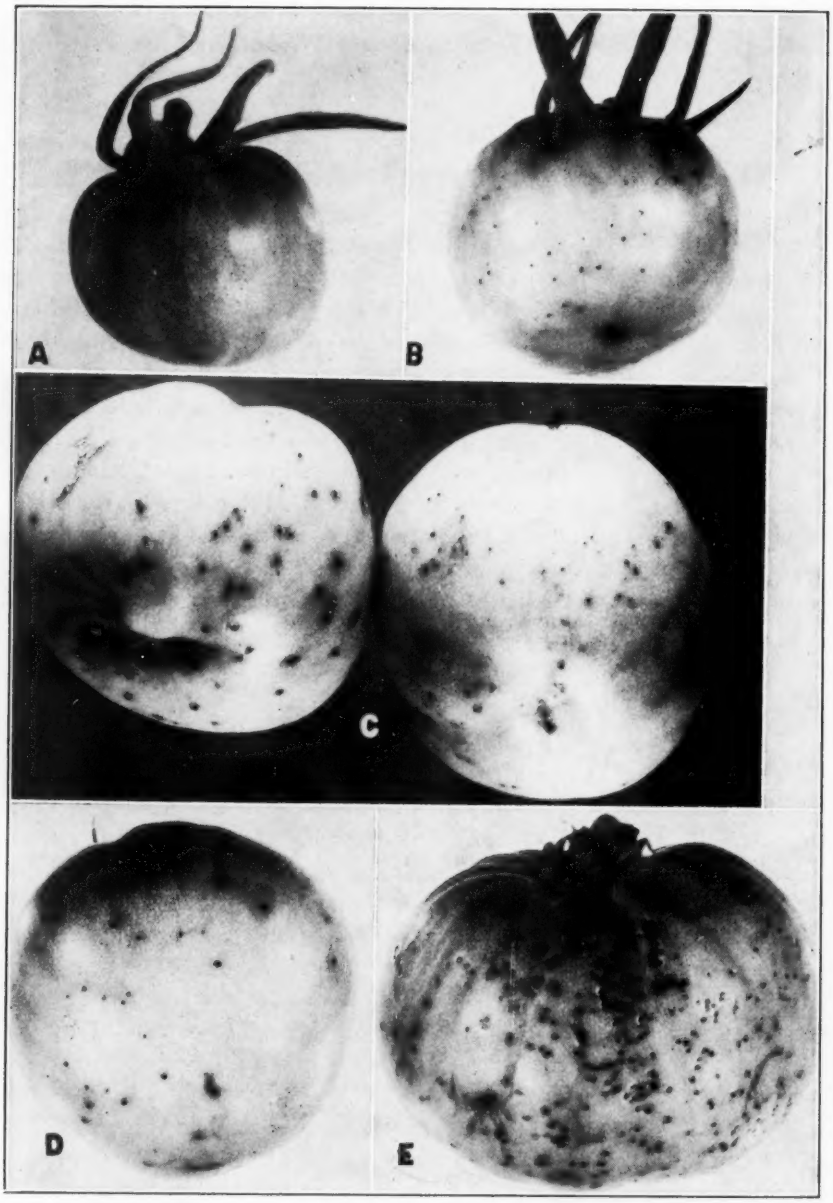

FIG. 1. Bacterial speck on tomato fruits. A. and B. Early stages. A, 7 days and B, 10 days after inoculation. C. Fruits in early stages of ripening showing darker green areas around the spots 15 days after inoculation. D and E, 21 days after inoculation. D. Scattering spots. E. Crowded spots.

D and E.) The spots are very slightly raised, flat and dark when first observable. (Fig. 1, A.) At this early stage, as well as when fully developed, they are round and flat with a clear-cut margin. As the fruit whitens before the pink stage, the tissue immediately surrounding the spots remains green but without clearly defined margins. (Fig. 1, C.)

The size of the spots depends on the age of the fruit when inoculated, those on very young fruit attaining the largest size. The epidermis remains unbroken. Lesions are, at first, very superficial. The epidermis is raised and separated from the underlying tissues by a dense mass of bacteria. Later, there is somewhat deeper penetration, with an attempt to cork off the intruder. The spot never penetrates so deeply as either bacterial spot or bacterial canker.

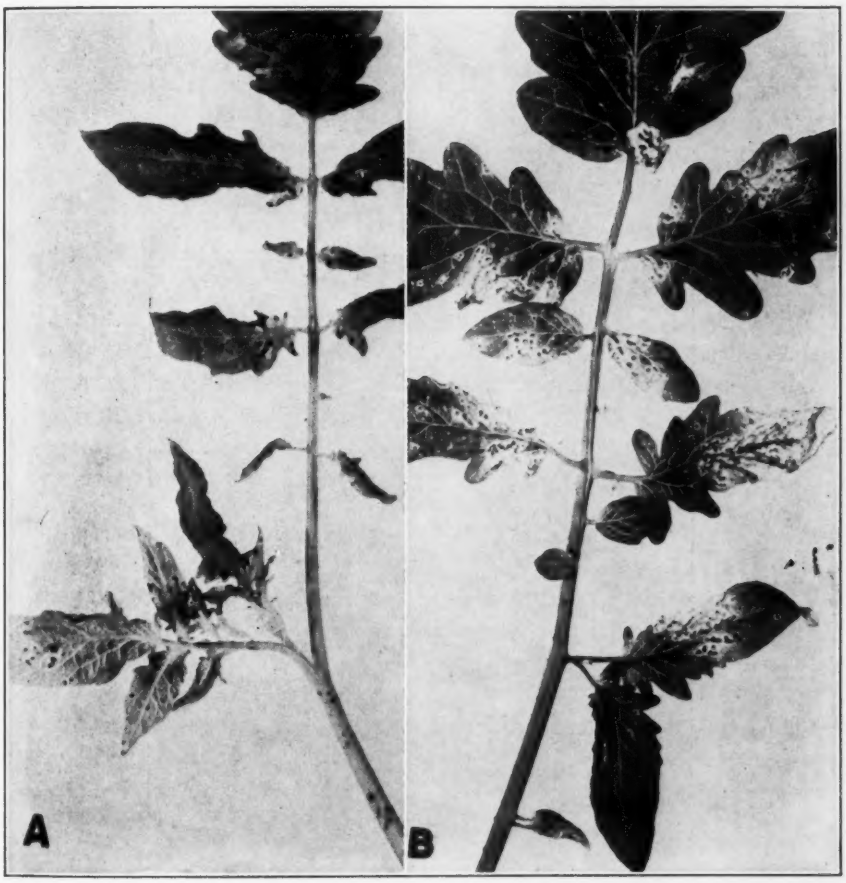

FIG. 2. Spotting on tomato stem and leaves inoculated with *Bact. punctulans*. A. 6 days after inoculation. B. 1 month after inoculation.

Lesions on the leaves are round, dark brown or black, without halo of any sort in early stages, and do not attain any larger size than the fruit spots. (Fig. 2, A.) They extend the entire thickness of the leaf, showing more conspicuously on the lower, paler surface. When close together spots coalesce into large irregular blotches and the surrounding tissues become yellow. (Fig. 2, B.) The petiole and petiolules also may be attacked.

Infections have been produced on stems, petioles, peduncles, pedicels, and sepals. In all these positions the spots tend to be oval rather than round, elongated with the long axis of the organ concerned. The peduncle

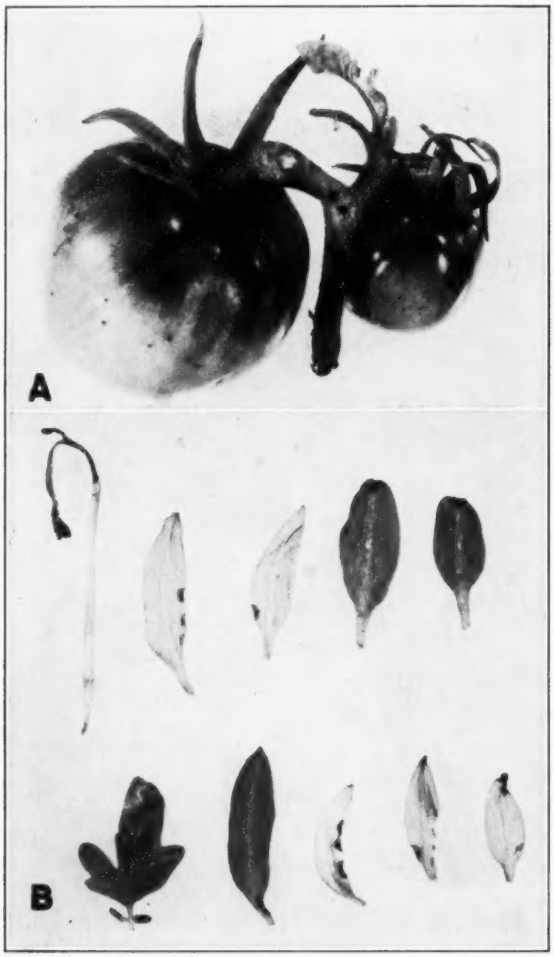

FIG. 3. A. Lesions on peduncle. B. Lesions on seedling leaves and on a seedling stem from inoculated seed.

seems especially susceptible. Here, lesions often coalesce to form large spots. (Fig. 3, A.)

INOCULATIONS

Inoculations on the fruit were made in two ways: 1, by swabbing lightly with a wad of cotton dipped in a water suspension of the organism; 2, by thrashing the fruit lightly with a leaf, simulating storm injury, then spraying with the water suspension. As with other bacterial spots of the tomato fruit, infection takes place through wounded hairs, there being no stomata present. Signs of infection are first observable after 5 days, as minute light brown points. These enlarge slowly. When very virulent strains were used on vigorous, young fruit, the minute infections in early stages oozed bacteria in such quantities as to show as white cirri. Only young fruits are susceptible.

Leaves and stems were inoculated by thrashing with a leaf followed by spraying with the inoculum. It appears that here, as well as on the fruits, the parasite enters through wounded hairs, for many more infections are obtained when the hairs are injured by thrashing than when the uninjured leaves and stems are sprayed and kept moist in inoculation chambers. Infections on seedlings have been obtained by planting clean seed in inoculated soil and also by planting inoculated seed in clean soil. Dark lesions appear on the cotyledons soon after they emerge, usually on the margins, but do not attain more than 1 mm. in diameter. Lesions also occur on the young stem and, in such cases, the whole top becomes involved, bends over, and blackens. (Fig. 3, B.)

Attempts were made to infect other cultivated plants. Seedling and well-grown eggplants, bell peppers and pimentos were inoculated by spraying the leaves and by swabbing young fruit, without obtaining any infections. Inoculations on bean, cucumber, and squash leaves also failed to produce infections.

THE ORGANISM

Morphology and Staining Reactions: Bact. punctulans is a rod varying from 1.3 to 2.5 μ long by 0.6 μ wide, measured on preparations from 24-hour pH 7.0 beef agar slants, occurring mostly as single rods or pairs. Long chains and filaments are also formed. The rods are motile by means of a tuft of several (as many as 7 were counted), polar flagella. Slender capsules were demonstrated by Ribbert's dahlia stain. No spores are formed. The organism is Gram-negative, not acid-fast, and stains readily with the usual bacterial stains.

CULTURAL CHARACTERS

Beef-infusion agar pH 7.0: Colonies on beef-agar plates are visible within 24 hours, and by the second day are 2 mm. wide, white, round, translucent,

and finely cross-hatched by oblique light. Later, they develop a more opaque white center and may reach a diameter of 9 mm., while the substratum becomes more or less green.[1] Occasional rough colonies appear on isolation plates. These are irregular in outline with a contoured or roughened surface and coarse cross-hatching. Platings from strains that have been on culture media for some time show more variations in colony type than platings direct from the plant. On slants the growth is moderate, spreading, flat, transparent, glistening, with slightly undulant margin. The medium becomes green.

Thaxter's potato agar: On this medium growth is abundant but not fluid; smooth, white, and opaque.

Beef-infusion broth pH 7.0: Clouding occurs within 24 hours and becomes heavy with membranous pellicle. In undisturbed cultures the pellicle is weighed down by a mass of white growth accumulating in the center. Green fluorescence develops after two or three days.

Potato cylinders: Growth is very moderate, inconspicuous, and transient. The substratum is slightly browned.

Synthetic media: Uschinsky's solution becomes heavily clouded with a membranous pellicle like that in beef broth. The medium quickly takes on a beautiful blue-green fluorescence. In Fermi's solution there are less clouding, a fluorescence more blue than green, and a heavy pellicle. Extremely weak but definite growth occurs in Cohn's solution.

PHYSIOLOGY

Liquefaction of gelatin. Liquefaction of beef gelatin at 18°–22° C. is napiform, beginning within 24 hours. Fifteen to 20 days are required for complete liquefaction. Abundant flocculent precipitate accumulates in the funnel. The medium becomes green.

Reactions in milk. Litmus milk begins to blue within 24 hours. Separation begins at the same time and in 5 days the semisolid curd occupies three-fourths to one-half of the culture. There is no banding. Reduction begins on the third to the fifth day and is complete within 2 weeks. The cream rim is reddened. The curd becomes firm in about 2 weeks. No peptonization occurs.

Hydrolysis of starch. Starch is not digested. Growth on potato cylinders is restricted and transient. On starch agar plates, flooded with iodine solution, after 7 days' growth, there is a slightly paler area immediately surrounding the growth bu no clear zone.

[1] Some strains are very strongly green fluorescent, others very weakly so, and some show no fluorescence in any medium. The nonfluorescent strains show also weaker virulence and are less vigorous in their reactions in culture media, especially litmus milk and gelatin.

Reduction of nitrates. There is strong nitrate reduction. Tests were made with the α-napthyl-amine-sulphanilic acid method, in cultures grown 10 days in nitrate beef broth.

Fermentation of sugars. Synthetic agar containing brom-cresol purple and 1 per cent of the following carbonhydrates was used: sucrose, dextrose, maltose, lactose, glycerine, and mannit. Slants were streaked from young broth cultures. No acid was formed from lactose or maltose. Acid reaction was evident in 24 hours from sucrose, dextrose, and mannit, and in 4 days from glycerine. After 6 days the agar was acid throughout with dextrose and in 10 days with sucrose, mannit, and glycerine. The same media, but without agar, were used in fermentation tubes. No gas was produced from any of the carbohydrates used. There was no growth evident in the closed end of any of the tubes, although the medium became acid in dextrose.

Relation to free oxygen. The organism is aerobic.

Optimum pH for growth. In a series in beef broth ranging from pH 5.0 to pH 9.0 clouding appeared within 24 hours in 6.0 to 8.7, best in 6.6 to 7.4, weak in 6.0 and in 7.6, and very weak in 8.3 and 8.7. After 48 hours the 5.4 showed growth. At the end of a week clouding was heavy in all from pH 5.4 (pH 7.0 at this date) to 7.6 (pH 8.1 at this date), moderate in 8.3, and weak in 8.7. All were green fluorescent from pH 7.4 to 8.7, most strongly so in 7.4 and 7.6.

Other physiological reactions. Bacterium punctulans does not form hydrogen sulphide or indol. Ammonia is formed slowly. Strips of filter paper dipped in Nessler's solution and suspended in 10-day-old beef-broth cultures did not show the ammonia reaction, while in 20-day cultures the reaction was good.

Temperature relations. The thermal death point is 51° C. The optimum temperature for growth is 23° to 25°. Growth is weak at 30°. At 35° and 37° beef-broth cultures not only did not grow but failed to cloud the medium when returned to room temperatures after 6 days at these temperatures. Slow growth with eventually good clouding occurs at 15° C. Clouding is visible in beef broth at 3° C. within 6 days.

Resistance to desiccation. The organism is very sensitive to drying. Preparations on cover glasses from 24-hour pH 7.0 beef bouillon, as well as from 3-day bouillon cultures, failed to give growth when dropped into bouillon after 48 hours' drying. One strain survived 9 days on three of ten covers placed in bouillon after that period, but required 8 days to show clouding.

BRIEF CHARACTERIZATION

Bacterium punctulans is a rod-shape bacterium, occurring singly, in pairs, and in chains, measuring 1.3–2.5 μ × 0.6 μ, motile by means of a tuft of polar flagella. It is Gram-negative and not acid-fast; forms thin, white

colonies with green fluorescence on beef agar; clouds bouillon heavily with a membranous pellicle, heavy in the center; no diastatic action; liquefies gelatin rapidly; forms acid without gas from sucrose, dextrose, glycerine, and mannit but not from lactose or maltose; reduces nitrates; blues, coagulates, and reduces litmus milk; is aerobic; produces ammonia but not hydrogen sulphide or indol; grows best in pH 6.6 to 7.4, is very sensitive to drying; optimum temperature 23°–25° C. Pathogenic to tomatoes.

SUMMARY

A disease of tomatoes designated as speck, heretofore confused with bacterial spot (*Bacterium vesicatorium*), is described. Speck has been found in Florida and in Maryland. It causes small, round, dark, superficial spots on tomato fruits. Stems and leaves also are readily infected. The bacteria enter through wounded hairs. The causal bacterium belongs to the green fluorescent group and is designated as **Bact. punctulans,** n. sp. Cultural and physiological characters of the organism are given.

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REACTION OF MAIZE SEEDLINGS TO GIBBERELLA SAUBINETII[1]

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(Accepted for publication Dec. 1, 1932)

The use of resistant varieties in controlling plant diseases caused by fungi is dependent upon the extent to which it is possible to obtain resistant varieties satisfactory also in other characteristics. A number of root-rotting organisms attack corn, and among these *Gibberella saubinetii* (Mont.) Sacc. has received considerable attention. Cooperative studies carried on by the Divisions of Plant Genetics and Plant Pathology have had as their aim the isolation of resistant selfed lines of corn and their recombination in crosses, to ascertain whether lines could be isolated that would remain resistant and whether such resistance was of practical importance in corn improvement.

The present report consists, for the most part, of a study of later generations from crosses reported previously by McIndoe.[3]

PREVIOUS STUDIES

A review of the previous investigations having a more or less direct bearing on the problem was made by McIndoe in 1931.[3] In brief, Dickson and coworkers[4] have shown that the extent of seedling blight caused by Gibberella is dependent largely upon soil temperature. After isolating resistant and susceptible lines of corn they learned also that susceptible strains were severely injured at temperatures below 24° C., while resistant strains were attacked below temperatures of 16° C. This led to the method of studying resistance and susceptibility of seedlings grown under controlled conditions in the greenhouse.

Dickson thought that the host plant was least susceptible when grown at temperatures most favorable to its development, *i.e.*, under conditions of normal metabolism; at temperatures above 24° C., the host plant would

[1] Cooperative studies between the Division of Agronomy and Plant Genetics and the Division of Plant Pathology and Botany. Paper No. 1148 of the Journal Series of the Minnesota Agricultural Experiment Station.

[2] Chief and Assistant Professor, Division of Agronomy and Plant Genetics, respectively, and Head, Section of Plant Pathology, Division of Plant Pathology and Botany.

[3] McIndoe, K. G. The inheritance of the reaction of maize to *Gibberella saubinetii*. Phytopath. 21: 615–639. 1931.

[4] Dickson, J. G. Influence of soil temperature and moisture on the development of the seedling blight of wheat and corn caused by *Gibberella saubinetii*. Jour. Agr. Res. 23: 837–870. 1923.

———. The relation of plant physiology and chemistry to the study of disease resistance in plants. Jour. Amer. Soc. Agron. 17: 676–695. 1925.

succeed in throwing off attacks of the pathogen, but, if susceptible lines were grown at lower temperatures, the abnormal metabolism permitted the development of the pathogen. Peterson, however, concluded that the resistance of corn seedlings to attacks of root-rotting organisms is dependent upon the ability of the seedlings to develop a functioning nodal root system before the mesocotyl is completely rotted.

Dickson and Holbert[5] concluded that first-generation crosses between resistant and susceptible strains of corn were susceptible at all temperatures, *i.e.*, that there is complete dominance of susceptibility.

McIndoe used the most resistant and susceptible lines isolated previously in the corn-breeding studies carried on at the Minnesota station. He studied also the reaction of a large number of F_1 crosses in comparison with that of the parental lines when grown under comparable controlled temperature conditions in the greenhouse. These crosses consisted of the F_1 combinations between long-time selfed lines, which were being used in learning the more desirable combinations. Conflicting results were obtained in the F_1 generations. Certain F_1 crosses were highly resistant, being more resistant than either parent; others were more susceptible than either parent, although, in general, there was little or no relationship between the reaction of the parents and of the F_1 crosses. It would seem from these results that the causes of resistance are very complex. McIndoe studied the reaction of F_3 lines selected at random and included in the study crosses between resistant × susceptible, susceptible × intermediate, and susceptible × susceptible parents. There appeared to be a somewhat higher degree of resistance in the F_3 lines from crosses, one of whose parents was classed as resistant, than in the F_3 lines from crosses between parents both classed as susceptible, although the type of segregation appeared relatively complex.

EXPERIMENTAL MATERIAL AND METHODS

The same strain of *Gibberella saubinetii* studied previously by McIndoe has been used also in these studies. As reported by McIndoe, the soil temperature rarely varied more than 2° C. about a mean temperature of 15° C.

Seed from the cultures to be tested was divided into 2 parts, one of which was treated with Semesan solution and planted in the controlled-temperature greenhouses to determine normal viability of the seed. The other part was inoculated with a pure culture of *Gibberella saubinetii* grown previously for about 10 days in a moist mixture of wheat and oat grains. The inoculum was prepared by adding tap water to the mixture, straining through cheesecloth, and standardizing by dilution, so that the concentration of

[5] Dickson, J. G., and J. R. Holbert. The influence of temperature upon the metabolism and expression of disease resistance in selfed lines of corn. Jour. Amer. Soc. Agron. **18**: 314–322. 1926.

spores was relatively uniform, the standard of comparison being 30–50 spores under the low-power field of the microscope.

The corn grains were planted in sand at uniform depth and the number of healthy plants was used as a criterion of the degree of resistance of the row. A correction was made on the basis of the germination obtained from the seed treated with Semesan Jr. in order to determine the extent of injury caused by the pure culture of Gibberella used in the studies.

The reliability of the method of study was determined by growing inoculated seed from each ear in 4 replications in 1930–31 and 3 in 1931–32, each test consisting of seeds planted in a single row in the greenhouse bench, the rows in each replicate series being randomized.

The host material consisted chiefly of later generations, F_4 and F_5, from the crosses reported by McIndoe. The studies were primarily of two sorts, correlation between reaction of rows in the different replications grown from seed of the same ears and interannual correlations to learn the extent to which manner of reaction was inherited under the conditions of the experiment.

The F_3 lines used by McIndoe consisted of F_3 progeny of random selections of F_2 selfed plants of the different crosses. F_4 progeny were grown and several ears were selfed in each line. The interannual F_3 to F_5 correlation coefficients reported consist of the relation between the seedling reaction of the progeny of the parent ear and the seedling reaction of the progeny of selfed ears of desirable plants of the following generation.

EXPERIMENTAL RESULTS

Because of the nature of the results it appears unnecessary to present them in great detail. The correlations between the reaction of replicates in 1930–31 are given in table 1. Crosses 43×46 and 43×47 are between resistant and susceptible lines, respectively, 49×50 between susceptible and intermediate parents, and 64×66 is the reciprocal type of cross, while both parents in the cross 58×60 were classed as susceptible.

The 5 crosses were studied separately, correlation coefficients being computed between the reaction of material grown in different replications.

All possible relationships were studied, *i.e.*, between replications 1 and 2, 1 and 3, 1 and 4, 2 and 3, 2 and 4, and between 3 and 4. All calculated coefficients were more than 3 times their probable errors, 13 of the 30 being over .5 and 17 less than .5. Considering the fact that only 10 seedlings were grown in each row, the extent of the relationship between different replicates seems rather satisfactory. When the average reaction between replications 1 and 2 was correlated with the results of the average of 3 and 4 all correlations were over .5, the smallest being $.5804 \pm .0437$ and the largest $.8425 \pm .0416$. These results appear rather satisfactory and in-

TABLE 1.—*Correlations between the reaction of replicates of F_4 progeny of crosses when inoculated with Gibberella saubinetii and grown under controlled temperatures. 1930–31*

Cross	Replicates 1 and 2	Replicates 1 and 3	Replicates 1 and 4
43 × 46	.6583 ± .0381	.5264 ± .0485	.4272 ± .0549
43 × 47	.2718 ± .0609	.3268 ± .0588	.4183 ± .0543
49 × 50	.5475 ± .0661	.5364 ± .0673	.6388 ± .0559
64 × 66	.4273 ± .1082	.3965 ± .1114	.6762 ± .0718
58 × 60	.8006 ± .0515	.7753 ± .0574	.6804 ± .0773

Cross	Replicates 2 and 3	Replicates 2 and 4	Replicates 3 and 4	(Av. 1 and 2) and (Av. 3 and 4)
43 × 46	.4424 ± .0540	.4085 ± .0559	.4427 ± .0539	.5812 ± .0445
43 × 47	.3844 ± .0561	.3567 ± .0574	.3822 ± .0562	.5804 ± .0437
49 × 50	.4576 ± .0746	.4813 ± .0726	.6841 ± .0503	.6675 ± .0544
64 × 66	.3444 ± .1166	.7892 ± .0499	.8720 ± .0317	.6641 ± .0740
58 × 60	.4407 ± .1158	.7693 ± .0588	.3603 ± .1252	.8425 ± .0416

dicate that the method of studying a short row grown from seed immersed in a water suspension of spores of *Gibberella saubinetii* is a fairly accurate means of determining the manner of reaction of seedling progeny of individual selfed ears.

Similar studies were made in the winter of 1931–32, with F_5 seedlings of the same crosses. Three replicates were used in this study and levels of significance determined by the use of Fisher's tables. In general, somewhat lower correlations were obtained than in 1930–31, although the calculated coefficients were for the most part significant. The extent of correlation ranged from + .270 to + .803. In the light of previous years' study it seems reasonable to conclude that the average of all three replications would show rather satisfactorily which of the F_5 lines were resistant and which susceptible.

TABLE 2.—*Correlations between the reaction of replicates of F_5 progeny of crosses when inoculated with Gibberella saubinetii and grown under controlled temperatures*

Crosses	Replicates 1 and 2	Replicates 1 and 3	Replicates 2 and 3	N	Approximate level of significance P = .01	P = .05
43 × 46	.430	.567	.397	83	.283	
43 × 47	.478	.803	.516	30	.449	
49 × 50, 58 × 60, and 64 × 66	.270	.361	.393	41	.393	.304

To be of value as a means of isolating resistant lines there should be a substantial relation between the reaction in different seasons: some lines should remain constantly resistant, while others should be consistently susceptible. Interannual correlation coefficients for the reaction between F_3 and F_4 as well as F_4 and F_5 are given in table 3. Studies are given also for selfed lines of Crosby and Golden Bantam.

TABLE 3.—*Interannual correlations of reaction of maize seedlings to seedling blight*

Nature of correlation	Years selfed	N	r	Approximate level of significance P = .01
43 × 47 (R × S)	F_3 and F_4	121	.095	.2540
43 × 46 (R × S)	F_3 and F_4	111	.074	.2540
49 × 50 (S × I)	F_3 and F_4	53	.092	.3541
58 × 60 (S × S)	F_3 and F_4	23	.312	.5368
64 × 66 (I × S)	F_3 and F_4	29	.133	.4487
43 × 47 (R × S)	F_4 and F_5	42	.055	.3932
43 × 46 (R × S)	F_4 and F_5	85	.205	.2830
(49 × 50), (58 × 60), and (64 × 66)	F_4 and F_5	43	.060	.3932
Crosby	3 and 4 yrs. selfed	40	.536	.3932
Golden Bantam	3 and 4 yrs. "	60	.280	.3248

In this study the reaction of F_3 seedlings of a particular selfed ear was correlated with the reaction of F_4 seedlings of selfed ears of the same progeny line. Similar methods were used to correlate F_4 and F_5. In the progeny of the various types of crosses none of the correlations are of significance. This leads to the conclusion that reaction of seedlings under controlled temperatures to attacks of *Gibberella saubinetii* cannot be used as a means of isolating resistant lines unless conditions are controlled to a greater extent than in the experiments reported here.

During 1930–32 a study was made of the interannual correlation between the reaction of 3-year selfed lines with that of the 4th-year selfed, using 40 lines of Crosby and 60 of the Golden Bantam. In this case both correlations were positive and that for the 40 Crosby lines was .536, with a level of significance for a P value of .01 of .3932. Most of these sweet-corn lines appear very uniform under normal field conditions, and the extent of homozygosis may explain why they gave a sensible relation between the reaction of 3rd- and 4th-generation selfed lines.

In the plant-breeding studies with corn, yielding ability of selfed lines is obtained by husking the ears of 10 plants and estimating the yield on a scale of 1 to 10. A considerable number of selfed lines of Golden Bantam

and Crosby sweet corn have been treated in this manner and their yield index correlated with reaction to *Gibberella saubinetii*. The results of this study are given in table 4, together with correlation between length of ear of the selfed plant and its seedling-blight reaction.

TABLE 4.—*Correlations between seedling reaction to Gibberella saubinetii and indices of plant vigor*

Characters correlated		N	r	Approximate level of significance P = .01
Gibberella reaction and ear length:				
Golden Bantam	5 years selfed	52	−.149	.322
" "	4 " "	100	.084	.254
Crosby	4 " "	49	.113	.358
Gibberella reaction and yield index of the parental line:				
Golden Bantam	4 years selfed	60	.285	.326
" "	3 " "	205	.048	.254
Crosby	4 " "	46	.046	.372
"	4 " "	50	.061	.358
"	3 " "	252	.0001	.254
Gibberella reaction and yield index of its progeny:				
Golden Bantam	3 years selfed	43	.029	.393
Crosby	3 " "	62	.048	.325

Of the 10 correlations given in table 4 only one between Gibberella reaction and yield index in 60 4-year selfed lines of Golden Bantam was of possible significance.

SUMMARY AND CONCLUSIONS

The manner of reaction of corn seedlings to attacks of *Gibberella saubinetii* was determined by inoculating seed with a pure culture of the organism and planting the seed in sand in a greenhouse where the soil temperatures were controlled within a range of approximately 2° C., an average soil temperature of 15° C. being maintained.

There was a sensible relation between the reaction of replicates when seedling progeny from the same ears were studied, indicating that the method was sufficiently accurate to be used as a means of isolating lines that might be consistently resistant or consistently susceptible.

Interannual correlations, expressing the extent to which there was an inheritance of manner of reaction to *Gibberella saubinetii*, were for the most part so small that it seems safe to conclude that the method is not reliable as a means of isolating lines that may be expected to behave in a consistent

manner. In other words, the progeny of individual ears were relatively constant in the extent of seedling blight, although inheritance was not apparently the main cause of uniformity of reaction in different replicates. It seems safe to conclude, therefore, that the conditions under which an ear develops determines in some way the manner of reaction of the progeny.

The results of these studies have made it apparent that a study of seedling blight in the manner here outlined is of little or no value in a corn-breeding program. This has led to the treatment of a part of the seed of each selfed ear with Semesan Jr. and the comparison of viability under field conditions with the progeny of nontreated seed. In future studies it is planned to inoculate a part of the seed of each selfed ear with Gibberella, and another part with Penicillium before planting in the field and compare the results with seed treated with Semesan Jr.

No significant relationship was obtained between reaction to Gibberella and indices of plant vigor.

INFLUENCE OF SPRAY APPLICATIONS ON AIR TEMPERATURES SURROUNDING SPRAYED POTATO PLANTS

H. R. Rosen

(Accepted for publication, Jan. 9, 1933)

For a number of years the writer has been attempting to determine the influence of such biologic and ecologic factors on the origin of tip and margin burn of Irish potato leaves as the relationship of leaf hoppers and flea beetles to such injury and the influence of light intensity, wind velocity, relative humidity, precipitation, amount of soil moisture, and air and soil temperatures.

Since two different types of etiologic agents have been postulated in the literature of potato tip and margin burn, one associated with the leaf hopper, *Empoasca fabae,* and the other with dry, hot weather, it seemed desirable, in view of the extreme prevalence of such injuries in Arkansas, to determine which of these factors was commonly involved in this State and to determine the efficacy of various control measures. This paper records precipitous drops in air temperature noted in conjunction with these studies.

Numerous investigators have found that Bordeaux mixture applied to Irish potatoes acted as a preventive for tip and margin burning, particularly against the type induced by leaf hoppers. Accordingly, this spray material among others has been used by the writer for several years with excellent results in controlling tip and margin burning in the northern, hilly part of Arkansas, and with little or no result up to the present in the southern, coastal plain part of the State.[1]

Indeed, any control of hopper injury obtained at Hope has been more than offset by the amount of spray injury encountered there and in marked contrast with that at the cooler, northern station at Fayetteville, where no such injury with Bordeaux sprays has occurred. In fact, marginal leaf burning has been so common at Hope in plots sprayed with Bordeaux mixture 4–4–50 and similar formulas, with symptoms very comparable to leafhopper injury, that, so far as concerns leaf injury, it has been difficult to distinguish sprayed from nonsprayed plants. These leaf injuries associated with spraying were absent or very slight in the first half of the potato-growing seasons and became more pronounced as the season advanced.

What is the reason for this discrepancy? An attempt to answer this question included a consideration of various injurious effects that might derive from Bordeaux mixture under certain climatic conditions. The

[1] The results here recorded were obtained at the Fruit and Truck Branch Experiment Station, Hope, Arkansas.

answer is not certain, but, in the course of analyzing the thermographic records of soil and air temperatures prevalent around sprayed and nonsprayed plants, the charts in almost every instance revealed very interesting drops in air temperatures occurring immediately after the application of the sprays, particularly during the latter half of the growing season. The thermographic records of nonsprayed plants representing the same intervals of time as the records of the sprayed ones show no such drops (Fig. 1).

Out of 23 sprayings conducted during a 3-year period, only 2 show no positive indications of immediate drops in temperature following the application. Table 1 records the changes that occurred in 2 of these years and presents the temperature, wind velocity, precipitation, and relative humidity prevalent on the respective dates of spray applications.

It will be observed in table 1 that no reductions in temperature were recorded in 2 instances and that all the other sprayings were accompanied

TABLE 1.—*Weather conditions at the time of spray applications and the drops in temperatures attending the spraying of potato plants*

Date and hour sprayed	Temperature at time of spraying	Wind velocity at 6 p. m.	Precipitation during 24 hours previous	Relative humidity at 6 p. m.	Drop in temperature
1930	*°C.*	*m. p. hr.*	*in.*	*per cent*	*°C.*
April 22, 4 p. m.	32.0	1.6	0	76	5.0
May 7, 4 p. m.	28.5	5.4	0.83	83	6.0
May 14, 8 a. m.	22.0	2.3	0	61	0
May 21, 5 p. m.	28.0	1.2	0	76	4.0
May 28, 1: 30 p. m.	30.5	1.2	0	83	2.5
June 4, 4 p. m.	32.5	2.3	0	63	4.5
June 11, 5: 45 p. m.	29.0	2.4	0	53	3.0
June 18, 4: 15 p. m.	37.0	1.5	0	60	7.0
1931					
April 23, 11 a. m.	20.0	1.7	0.45	77	drop not clear (ink spot)
April 30, 3 p. m.	22.5	2.6	0	90	0 (rain immediately after)
May 7, 11 a. m.	21.0	3.1	0	64	4.0
May 14, 11 a. m.	27.0	2.2	0	50	5.0
May 21, 9: 30 a. m.	22.5	3.1	0	55	4.0
May 28, 8 a. m.	25.0	2.1	0	57	1.5
June 4, 2 p. m.	38.0	2.7	0	46	8.0
June 11, 8 a. m.	26.0	3.4	0.01	64	2.0
June 20, 10: 30 a. m.	34.0	2.7	0	71	5.5
June 25, 4: 30 p. m.	32.5	2.3	0	91	3.0

by a decided drop, varying in amount at different times. Unfortunately, the relative humidity and wind velocity were measured, not at the time of spraying but at 6 p. m. of the same day. Since these may be expected to vary considerably in any one day, the data on these 2 factors are not so satisfactory as they might otherwise be. It is to be noted that on May 14, when no drop was recorded, the spraying occurred at 8 a. m., when the air temperature (22.0° C.) was relatively low and on the other occasion, April 30, 1931, when rain fell immediately after the spray was applied. As may be expected, the greatest temperature drops occurred when the spraying was conducted near midday or in the afternoon, the largest drop being recorded at 2 p. m., June 4, 1931, when the relative humidity was very low and the temperature very high.

A clearer idea of the nature of these temperature drops may be gained by observing 2 air-temperature graphs of sprayed and nonsprayed plants (Fig. 1).

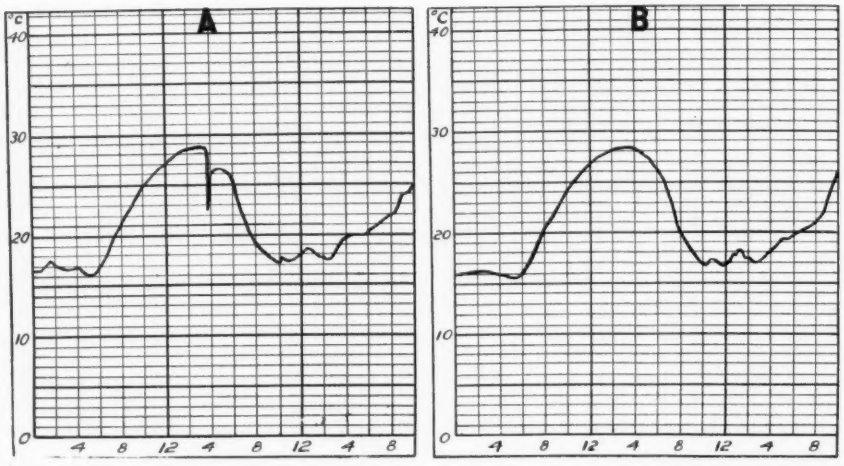

FIG. 1. A. Air temperatures existing around sprayed potato plants at Hope, Arkansas, from midnight, May 6, to midday May 8, 1930. Note sudden and precipitous drop in temperature shortly before 4 p. m. on May 7, when Bordeaux spray was applied. B. Air temperatures existing around nonsprayed potato plants at the same time and place as shown in A.

The temperature drop shown in figure 1, A, occurred, according to the records, shortly before 4 p. m. on May 7, 1930, immediately after the spray had been applied. The thermograph record shows a precipitous drop of 6.0° C. (12.6° F.) immediately followed by a perpendicular upward curve, coinciding almost exactly with the downward curve. The control plants (Fig. 1, B) showed no such abrupt change in temperature.

The thermograph instruments enclosed within housings comparable to those used by the United States Weather Bureau were placed at levels representing approximately the medium height of the potato plants, one between two sprayed rows and the other between two nonsprayed rows. The rows in each instance were 3 feet apart, and the temperature-sensitive portion of each instrument was, accordingly, about 18 inches from the center of each row. With the foliage extending about a foot outward from each side of the middles, the instruments were in excellent positions to record temperature changes occurring around the plants. However, considering the width of the instrument housing and its possible insulating effect, it is conceivable that greater temperature changes may have occurred immediately around the plants than were shown in the automatically recorded charts. This is particularly likely to be the case when the changes are of such short duration as shown in figure 1, A.

For many years it has been known that spraying various kinds of plants during the warm hours of the day may be attended by injury to foliage and fruit. The factors that are responsible for such injury are, however, obscure and, in many cases, unknown. Of course, the burning action of a drop of liquid, in functioning as a convex lens and centering the beams of light, is well known. Likewise, the intolerance of certain plants to some spray materials has been noted by numerous observers. It also is known that certain spray materials, such as Bordeaux mixture and lime sulphur, may cause foliage injury and fruit russeting or burning under some weather conditions and not under others. That Bordeaux mixture will often cause a russeting of apple fruit when applied in the early part of the growing season and produce little or no injury thereafter is well known. Why this is so is not clear.

As far as potato plants are concerned, it has been definitely established that a Bordeaux spray increases the rate of transpiration (1, 3, 4, 5, 6, 7, 8). What is responsible for the rapid and precipitous drops in temperature when this spray is applied, especially during the warm hours of the day? The answer probably is to be found in the evaporating power of the warm, dry air. The transformation of energy occasioned by such evaporation evidently results in a rapid lowering of temperature.

Thus, two different phenomena may attend the spraying of potato plants and probably other plants as well, one, a quick lowering of air temperatures around the sprayed parts and the other, an increase in the rate of transpiration. It has not been shown, however, that the two occur simultaneously. It is conceivable that other liquid sprays may bring forth similar responses. Investigators as a whole have not associated any injury with increased transpiration. On the contrary, they have found that, despite such increases, the potato foliage does not suffer, even during dry seasons, and the yield of tubers is often increased, even in years when the control

of late blight and other foliar disturbances are not involved (2, 9). What influence may sudden changes of air temperature have on potato plants? Can changes of such short duration as here shown have anything to do with the spray injury that has been noted at Hope, Arkansas, for 4 successive years?

Briefly summarizing, while effective control of tip and margin burning of potato leaves has been obtained by the use of Bordeaux sprays in the northern part of Arkansas, no satisfactory control with this material has been accomplished in the southern part during a 4-year period. In attempting to determine the cause of this discrepancy and to find an explanation for the spray injury, which that material has engendered in the southern portion of the State, a study has been made of various climatic factors existing around sprayed and unsprayed plants. It has been found that spraying in most instances is accompanied by sudden and precipitous drops in air temperature around sprayed plants, the drops varying from 1.5° to 8.0° C. (3.1 to 16.8° F.). What influence such abrupt temperature changes may exert on the health of the plant remains unknown. It is quite obvious, however, that in the warmer sections of the State, with prevailingly high temperatures during the latter half of the potato-growing season, temperature drops attending spraying would be much greater than in the cooler sections. Does this suggest that midsummer spraying of various kinds of plants in the warmer parts of America is attended by greater risks in so-called spray injury than in the cooler parts?

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RESISTANCE OF THE CURRENT SEASON'S SHOOTS OF PINUS MONTICOLA TO INFECTION BY CRONARTIUM RIBICOLA[1]

H. G. LACHMUND

(Accepted for publication November 3, 1932)

In studies of the blister rust (*Cronartium ribicola* Fischer) on western white pine, *Pinus monticola* Dougl., (3), the writer found that the cankers resulting from any year's sporidial infection were distributed in the following characteristic pattern on the different years' internodes exposed to the infection.

	Growth[2] *affected*	*Number of cankers*	
a	Growth of the season of infection	relatively few	
b	Growth 1 year older than *a*	greatest number	large majority
c	Growth 2 years older than *a*	considerable number	
d	Growth 3 years older than *a*	relatively few	
e	Growth 4 years older than *a*	very few	
f	Growth 5 years older than *a*	exceptional	
g	Growth 6 years older than *a*	none	
h	Growth 7 years older than *a*	none	

Tabulation, totalling approximately 6,000 cankers, gave about 10 per cent of the cankers on *a* growth, 53 per cent on *b*, 31 per cent on *c*, 5 per cent on *d*, less than 1 per cent on *e*, a trace on *f*, and none on older growths.

Cronartium ribicola infects its aecial hosts through the needles (1) on which it enters through the stomata and grows down the vascular bundles to the bark. In *Pinus monticola* it generally undergoes an incubation period of 2 years' duration before becoming apparent in the bark. Part of this period is required for the rust to develop in the needle and grow down from the infection point to the bark. The length of time required for this in *P. monticola* is not known. In *P. strobus* (1, p. 455) it appears to range from 2 to as high as 10 or 12 months and, in general, to be a matter of over 6 months.

Obviously, then, other conditions being equal, the number of cankers that develop on any year's growth as the result of infection in a given

[1] The writer was assisted by J. L. Mielke, C. N. Partington, W. V. Benedict, T. S. Buchanan, J. W. Kimmey, A. A. McCready, and W. F. Cummins in the collection of the basic data of this paper.

[2] The term "growth" is used to designate the internodes of a given year.

season should depend primarily on the quantity of needles on that growth and the length of time they are held following infection. Spaulding (6) has used similar premises for a partial explanation of differences in the susceptibility to the rust of different species of white pine.

In all growing trees there is normally a multiplication of twigs and branches and a progressive increase in foliage production each year to provide for the increased nutrition requirements of the tree as it increases in size. Each succeeding season's crop of shoots, therefore, has normally a greater total length, bark area, and production of foliage than that of the preceding season. Thus, from the standpoint of quantity alone, the chances of infection should be greatest on *a* growth and progessively smaller, the older the growth. These differences are very greatly and rapidly accentuated by the dropping of the leaves.

In the Pacific Northwest[3] (3) the needles of any season on *Pinus monticola* are regularly held for at least 3 full growing seasons. Part of them drop at the end of their third season, and most of the remainder fall at the end of the fourth season. The small portion left over are generally cast at the end of the fifth season, although, exceptionally, one or two needle bundles may persist as much as 8 seasons.

Infection of the pines in this region as elsewhere usually occurs in late summer and early fall. Figure 1 represents diagrammatically the normal retention of the needles at this time and the distribution of the resultant cankers, as shown by the tabulations already referred to above, in the second season following that of infection, when the cankers normally appear.

The reason for the negligible representations of cankers on *e* and older growths is immediately apparent. There are scarcely any needles on such growth at the time of infection, and such needles as are present normally fall at the end of the season or only shortly following the occurrence of infection. The relatively small representation of cankers on *d* growth is explained by the fact that such growth has already lost part of its needles and loses most of those remaining upon it at the end of the infection season. The smaller representation on *c* growth than on *b* growth may be explained on a similar basis. *C* growth loses part of its needles at the end of the infection season and most of the remainder one year later, whereas *b* growth holds all of its needles for at least a full year following infection and the majority of them for 2 years. Thus part of the individual infections on the needles of *c* growth may be cast with the needles at the end of the season before the fungus reaches the bark, while most of the infections on the needles of *b* growth should have ample time to gain entrance.

[3] Pacific Northwest as used in this paper includes southern British Columbia, Washington, Oregon, Idaho, and western Montana.

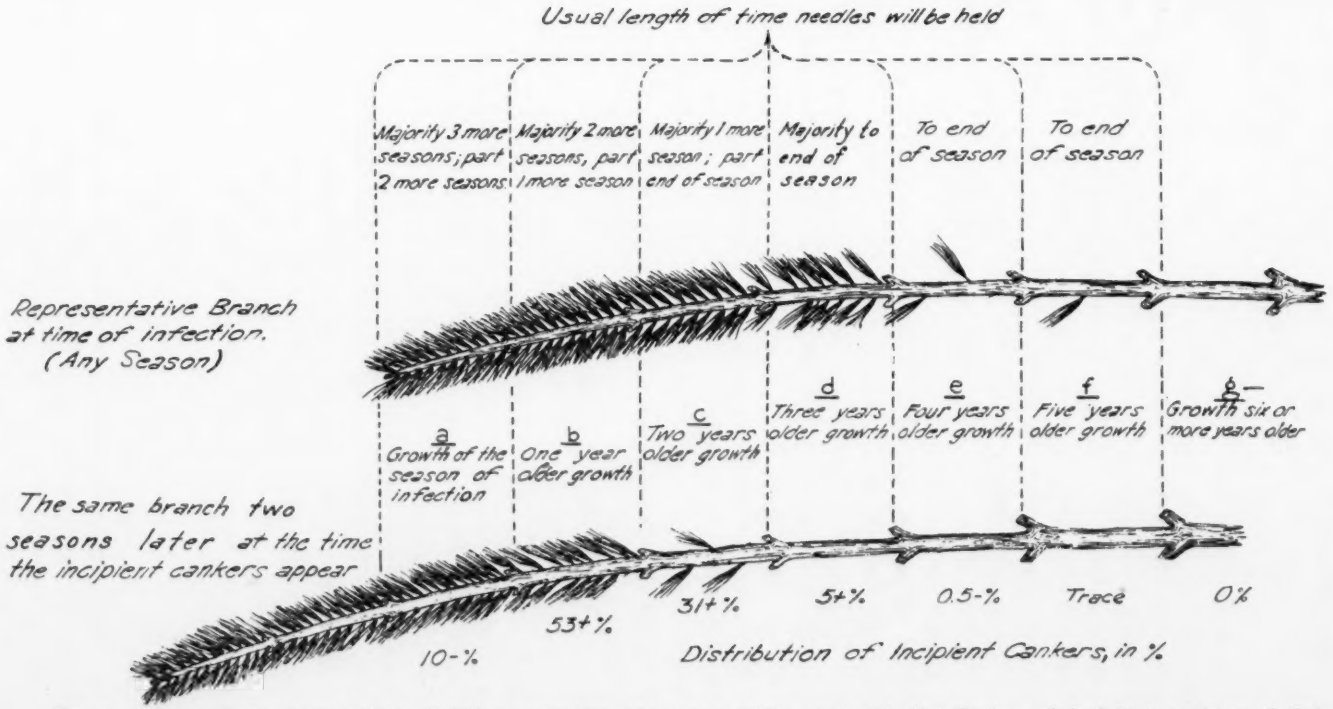
Usual length of time needles will be held
Majority 3 more seasons; part 2 more seasons
Majority 2 more seasons, part 1 more season
Majority 1 more season; part end of season
Majority to end of season
To end of season
To end of season
Representative Branch at time of infection. (Any Season)
a Growth of the season of infection
b One year older growth
c Two years older growth
d Three years older growth
e Four years older growth
f Five years older growth
g Growth six or more years older
The same branch two seasons later at the time the incipient cankers appear
10-%
53+%
31+%
5+%
0.5-%
Trace
0%
Distribution of Incipient Cankers, in %

FIG. 1. Diagrammatic presentation of foliage retention of western white pine and distribution of incipient cankers of *Cronartium ribicola* on internodes of different ages.

On this basis *a* growth, which not only has a much greater number of needles but holds them a full year longer than *b* growth, should have a greater proportion of the cankers than *b* growth. Yet the percentage on *a* growth is on the average only a fifth of that on *b* growth and a third of that on *c* growth.

There appear to be no differences in the position of the needles on the tree, their arrangement on the branches or in the fascicles, their size at the time of infection, or in microclimatic conditions surrounding infection that could offset the superiority in the number and retention of needles on *a* growth to a degree sufficient to account for the inferiority in the number of cankers upon it as compared with *b* and *c* growths. Likewise, there is no evidence of any physiological barrier in the young bark tissues of *a* growth to the entrance of the mycelium from the needles. Indeed, comparing stems of equal size, the most vigorous canker growth is found on the youngest, thriftiest stems. Consequently, since especial vigor encourages the growth of the mycelium and since vigor is greater, the younger the shoot, it would not appear that there should be any physiological barrier in the bark of *a* growth that would prevent entrance of the mycelium, once the latter had reached the base of the needle. Direct infection of the bark may be eliminated as an explanation, for it is already established that this is not an important method of infection (1, p. 441; 5, pp. 26, 27), and if it were, the conditions for it would be primarily favorable on *a* growth (1, p. 441; 8).

The resistance indicated by the relative scarcity of cankers on *a* growth, therefore, evidently centers in the needles of this growth. Whether it arises as a physiological reaction of the young tissues or from differences in the behavior of the stomata, as in the case of the resistance of certain wheat varieties to stem rust (*Puccinia graminis tritici* Eriks. and Henn.) (2), or in some other way, is not known.

Regardless of where or how this resistance in *a* growth originates, it is clear that it is a function of age. That it persists through the period in which important infection may take place during the first season is also evident, for the number of cankers resulting from any season's infection on the growth that originated in the year of infection (*a* growth) has never been more than a third of the number resulting on the growth a year older (*b* growth).

The proportion of the cankers on *a* compared with the number on *b* has varied from as low as one-twentieth to one-third. The most probable explanation for this wide variation would appear to be that the resistance in *a* needles decreases rather rapidly following their formation, while the susceptibility in *b* needles remains relatively constant. It would necessarily follow that where the sporidial infection wave occurred early in the season

the proportion of cankers resulting on *a* would be relatively small, and where the infection occurred late in the season the proportion would be comparatively large.

Whether the resistance persists to any degree into the second season or later is not known. The solution of this problem will require artificial infection experiments.

The resistance in the current shoots of *Pinus monticola* is in direct contrast to the evidence in this connection for *P. strobus* L. On this species the needles of *a* growth appear to be quite as susceptible as those on *b* growth (9, p. 507) and *a* growth, as might be expected from its superiority in number and retention of needles, generally has the majority of the cankers (4, p. 479; 7, p. 38).

SUMMARY

Of the cankers resulting from any season's infection of blister rust on *Pinus monticola* relatively few are found on growth (internodes) formed the season of infection. Yet the needles, through which infection occurs on the pines (1), are not only more numerous but are held a year longer on this growth than on any older growth and for this reason should provide better opportunities for the subsequent entrance of the individual infections. Conditions governing the occurrence of infection, such as exposure to sporidia, humidity, etc., should be essentially as favorable on the season's needles as on the older needles. Observations indicate that conditions for the development of the fungus, once it is established in the bark, should be equally, if not more, favorable on the growth formed in the infection season than on growth one year older. But the number of the cankers that develop on the infection season's growth averages only a fifth of the number that develop on the one-year older growth and a third of the number that develop on growth 2 years older. The small proportion of cankers resulting on the growth of the season of infection is explained on the basis that its needles are resistant to infection. The resistance probably declines very rapidly during the first growing season and does not extend to any degree into the second growing season.

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PHYTOPATHOLOGICAL NOTE

The Spermatia of Corn Rust, Puccinia sorghi.[1]—Since the discovery by Craigie in 1927 of heterothallism in rusts and the function of rust spermogonia (pycnia) it has been known that spermatia (pycniospores) of one sex when brought to an infection of opposite sex can initiate there the sporophyte generation of the rust and lead to the development of fertile aecia. Several workers since then have attempted to learn just how the spermatia function, and Craigie has depicted the union of spermatia and haploid hyphae in *Puccinia helianthi* Schw. Much, however, still remains to be learned.

Puccinia sorghi Schw. has proved favorable material for such a study. It is a heterothallic, long-cycle, heteroecious rust with uredia and telia on corn and spermogonia and aecia on species of Oxalis.

Oxalis plants for this study were grown in 6-inch pots on an outdoor balcony and inoculated there with the rust. When the infections matured, the spermogonial exudate was transferred back and forth among the infections, the attempt being made to bring exudate to each infection from several others. In order to maintain a saturated atmosphere about the plants, a battery jar was lined with wet paper towelling and sprayed full of mist with an atomizer; the plants themselves were sprayed lightly; the inverted jar was set down over the plants; more wet towelling was wrapped around the joining of battery jar and pot-rim; and the whole was set in a partly shaded place. At short intervals infected material was removed from the plants; part of it was fixed, and the rest used for making freehand sections of the living material.

Figure 1, A, represents semidiagrammatically a cross section of the infected Oxalis leaf with intercellular mycelium and open spermogonia at *a* and *b*.

In figure 1, B and E, are sketches from the living material showing paraphyses with spermatia attached. In B is a brush of paraphyses with the outer line of the epidermis at *c*. A paraphysis may carry only 2 or 3 spermatia (Fig. 1, B, *a*, *b;* C, *c*) or may be crowded with them (Fig. 1, D, *a*). The spermatia may be applied closely to the paraphysis (Fig. 1, B, *b;* E, *b*) or may be at some distance and connected by a slender hypha (Fig. 1, C, *a;* E, *a*). It is not always possible to be sure how many of these

[1] Cooperative investigations between the California Agricultural Experiment Station and the Division of Cereal Crops and Diseases, Bureau of Plant Industry, U. S. Department of Agriculture.

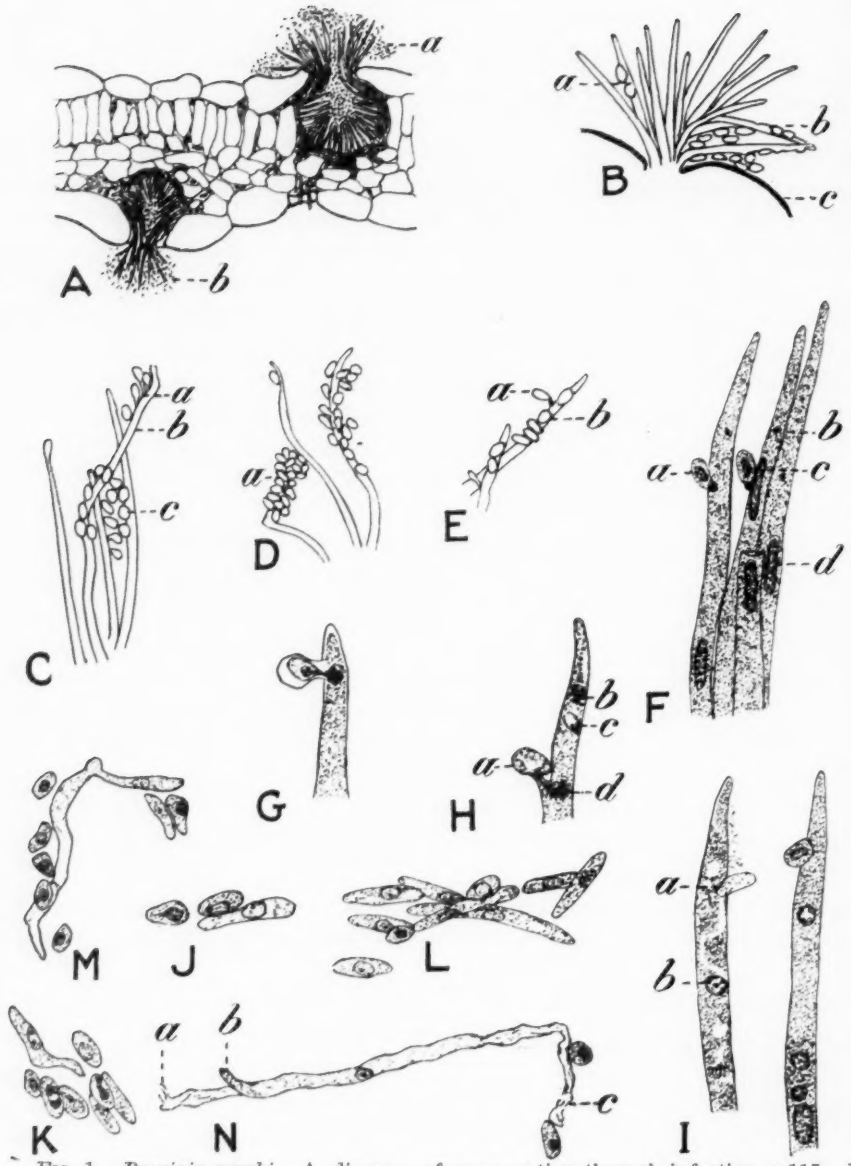

FIG. 1. *Puccinia sorghi*. A, diagram of cross section through infection, ×115. B to E, sketches from living material showing paraphyses with attached spermatia. Magnification not measured. F to I, stages of fusion of spermatium with paraphysis, ×1400. J to N, stages of germination of spermatia, ×1400.

spermatia are really attached to a paraphysis and how many are loosely adherent. As a precaution, before making the sketch, the cover glass was jogged and tapped repeatedly to free any loose spermatia. In some cases

the spermatia are certainly attached, for, by watching them under the microscope, spermatia like those at C, *a*, can be seen swinging back and forth when the cover glass is tapped.

In fixed and stained material the ordinary paraphysis is seen as a straight, rather stiff-looking cell, tapering at the tip. It contains a single nucleus, often somewhat elongated (Fig. 1, F, *d*) and dense cytoplasm sometimes bearing one or more dark-staining granules (Fig. 1, F, *b*).

A spermogonium forms new paraphyses as it grows older. When a plant has been in a saturated atmosphere for several hours, a newly emerging paraphysis may take on a different character. It grows more like an internal hypha in the saturated air of the intercellular spaces of the leaf. It is usually slender (Fig. 1, C, *b*), does not taper at the tip, follows an irregular course, and may branch. So far as noted, it is nonseptate.

Over 95 per cent of the spermatia attached to paraphyses wash off on the way to the completed slide. Enough remain to furnish cytologic details. In figure 1, F, 2 spermatia, *a* and *c*, have each secreted a small mass of dark-staining matter at the point of attachment to the paraphysis but have not started to enter. In figure 1, G and H, is an open passageway between spermatium and paraphysis. In G, the nucleus is moving in, and in H, *a*, *d*, transit is just completed. At *b* and *c* in figure 1, H, are what appear to be 2 other nuclei, and by focussing above the plane of the drawing, remnants of spermatia (not drawn) are found. In figure 1, I, is the remnant of a spermatium at *a;* at *b* is the introduced nucleus, now more expanded.

The irregular paraphyses formed under humid conditions also serve to receive spermatial nuclei (Fig. 1, C, *a;* D, *a*).

Under these excessively humid conditions and in the presence of mixed exudate, the spermatia on the leaf surface germinate in great numbers. Figure 1, J to N, shows details of germination of spermatia (×1400). The spermatium, at first elliptical, becomes cigar-shape (Fig. 1, J, K, L). On further growth, it may become more irregular (Fig. 1, M, N). The germ tube wall is very thin, and the contents stain lightly. So far as noted, it remains uninucleate and nonseptate. In figure 1, N, the germ tube started at *c*, grew to *a*, apparently met adverse conditions and the cytoplasm retreated and took a fresh start at *b*. Ordinarily, the germ tube remains unbranched.

Germinating spermatia are found also in mixed exudate at spermogonia. Their study has just been begun, but there are some indications that when paraphyses are young and vigorous, the spermatia fuse directly with them but when the paraphyses are old and decadent, the spermatia germinate and grow down through the ostiole into the spermogonium.

A more extended study of corn rust is in progress.—RUTH F. ALLEN, University of California, Berkeley, California.

BOOK REVIEW

Les Maladies à Ultravirus des Plantes. By Marie-Antoinette Beauverie. 175 pp., 8 pl. Reprinted from *Annales du Service Botanique et Agronomique de Tunisie.* Vol. 9, 1932. *Desvignes, Lyon.* 30 fr.

In summarizing the literature on the virus diseases of plants the author has faced a task of considerable magnitude and also of some difficulty, since the past ten years have witnessed an enormous increase both in the literature and in the complexity of the problem. Any attempt to condense this material into a paper of reasonable length is likely to result in occasional omissions and also in certain difficulties in the handling of conflicting evidence. The present work is not wholly free from such difficulties, but in view of the many discrepancies that appear in the literature itself, such omissions are not easily avoided. The very complexity of the problem, however, makes a work of this sort the more valuable, since it serves to bring the entire field of plant-virus research into at least an approximate focus. Mlle. Beauverie has accomplished this in very satisfactory fashion.

In taking up the subject the author has first defined the plant viruses on a basis of their characteristics, namely, filterability, ultramicroscopic nature of the causal agent, differentiation from filterable bacteria, biotropism, symptoms, and methods of transmission. Following this is a list of host plants based on the literature up to 1931 and a discussion of the wide-spread occurrence and the importance of control of these diseases. Chapter II deals with the technique of the study of the virus in extracted plant juices and furnishes a rather full discussion of methods of filtration and ultra-filtration as well as the effect of physical factors on the filtration process. It also covers the reactions of the viruses to heat, desiccation, dilution, adsorption, and other physical agencies, and the effect of chemicals and the possibilities of virus attenuation. The material is treated concisely and adequately and the discussion will prove of value to those who are interested in these phases of the problem. The third chapter presents the various theories regarding the nature of the virus and discusses the evidence on which these theories are based. Chapter IV gives a brief discussion of the distinction between infectious and noninfectious chloroses, and Chapter V deals rather fully with the pathological histology and cytology of plants affected by virus diseases. The material in this latter chapter is treated very satisfactorily, although it omits the work of Hoggan on the inclusion bodies associated with the different viruses affecting solanaceous plants and the value of such inclusions in the classification of certain of these viruses. Chapter VI takes up the transmission of plant viruses by insects and by artificial inoculation and the importance of soil, seed, and wild host plants

as sources of infection. This section might well have contained a more complete list of references on selective transmission by aphids in the case of certain combined virus infections, but it furnishes an excellent presentation of our knowledge of the transmission of these diseases. The same chapter deals with the variations in symptoms produced by a single virus on various hosts and the masking and alteration of symptoms that result from changes in environment. The methods by which the virus is thought to traverse the plant are also discussed.

Chapter VII describes the virus diseases affecting plants of economic importance, about one-third of the space being devoted to the virus diseases of the potato. This section has suffered somewhat from the condensed nature of the descriptions but it is generally adequate in its treatment. The discussion of tobacco mosaic, however, omits direct reference to the outstanding work of James Johnson on the virus diseases of his host and leaves the impression that the tobacco mosaics are due to a single virus. The statement that tobacco mosaic is transmitted by various aphids is also subject to qualification as a result of the work of Hoggan. Wheat mosaic and rosette are treated as distinct diseases, although the work of McKinney appears to have established the identity of these diseases. In making the above criticisms, however, it should be noted that each chapter is followed by a list of papers relating to the subject under discussion and that the omissions mentioned are generally included in these citations. Chapter VIII discusses the analysis of virus complexes by means of differential hosts, filtration, varying incubation periods, and selective transmission by aphids, while the concluding chapter outlines the analogies that exist between the plant and animal viruses. Following this is a bibliography of 762 titles, which is conveniently arranged and apparently very free from errors. Such a list of literature will prove of great value to all those interested in the subject, since there has been no previous compilation on such a comprehensive scale. The text also is admirably arranged for reference purposes, and any one who has reason to consult Mlle. Beauverie's work will agree that she has made a contribution of distinct value.—S. P. DOOLITTLE, Bureau of Plant Industry, Washington, D. C.

REPORT OF THE SEVENTEENTH ANNUAL MEETING OF THE PACIFIC DIVISION OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY

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The annual meetings of the Pacific Division of The American Phytopathological Society were held jointly with those of the Pacific Division of the Botanical Society of America at the University of Utah, in Salt Lake City, June 14–15, 1933. The meetings were held in conjunction with those of the Pacific Division of the American Association for the Advancement of Science and Affiliated Societies.

The effect of the world-wide depression was reflected in the poor attendance at the Salt Lake meetings, as a whole, but the meetings of the botanists and the phytopathologists were well attended, all things considered. Three half-day sessions were held, and as many as 50 members of the 2 societies were in attendance. The joint meetings in themselves proved an interesting variation from the usual routine, and the papers presented evoked considerable enthusiastic discussion. Abstracts of the papers concerned with plant pathology follow.

B. A. RUDOLPH, *Secretary-Treasurer*

ABSTRACTS

The dieback form of tomato streak.—M. SHAPOVALOV.

The term "streak" is a group name. Besides the combination streak, other forms have been reported. A very destructive form, locally known as "dieback," is prevalent on the Pacific Coast. The infection in some fields reaches 100 per cent. Affected plants, as a rule, become worthless. New shoots die back several inches and mosaic symptoms are characteristically absent, which is not the case with the combination streak. "Bronzing" is frequently seen.

Transmission of dieback by swabbing infective juices on healthy leaves is very difficult, whereas combination streak, under the same conditions, gave 100 per cent of infection. As revealed by biological differentiation tests, dieback virus does not contain either the green-tobacco or the latent potato virus. Of the 53 inoculated *Nicotiana tabacum* plants none developed symptoms, and retransfers to tomatoes gave negative results. Symptoms typical of the latent virus failed to develop on *N. glutinosa*. *Datura stramonium* occasionally developed mosaic without local lesions but with a tendency to leaf puckering; the inoculation of tomatoes with the juice from these Daturas failed to produce any disease.

Dieback symptomatologically is similar to "spotted wilt." The insect vector of dieback has not yet been discovered.

Some improvements in auto-irrigator apparatus.—L. A. RICHARDS and H. L. BLOOD.

To overcome the difficulty of preventing air leaks in prevailing types of two-piece double-wall irrigator pots a one-piece double-wall pot was constructed. Pots may be set

up for operation in groups by connecting them to a ¾-inch distributing pipe connected to an overhead reservoir that draws water from a constant-level supply reservoir through a connecting supply line. The air, which might leak into the system, passes to the overhead reservoir and may be removed with an aspirator pump. Single pots may be operated by equipping them with a supply tube leading into a supply reservoir and a modified thistle-tube reservoir on the respective arms of a ¾-inch "T" leading to the pot. The tension of the water in the pots is controlled by varying the water head or the amount of mercury inserted in the supply line. The capillary conductivity of the soil, the capillary conductivity of the porous-pot wall, the proportion of the soil to the water-supplying surface, and the rates of evaporation and of transpiration influence the precision of control obtainable with this system. The double-wall porous pot and modifications appear to have wide application in plant-disease investigations.

Some inoculations with Dothiorella ribis.—C. O. SMITH.

Inoculations through a wound on woody stems of plants with strains from the walnut, *Juglans regia*, and from the avocado, *Persea americana*, indicate that 25 species are susceptible and have lesions with a radius of 5 to 70 mm. The species successfully inoculated are as follows: *Citrus aurantium, C. grandis, C. limonia, C. sinensis* (most resistant); *Juglans regia, J. californica, J. Hindsii, J. sieboldiana; Carya pecan.* In the Rosaceae: *Cydonia oblonga; Eriobotrya japonica;* Rubus mammoth blackberry; Rosa Lady Hillingdon and Maman Cochet; *Prunus communis, P. domestica, P. avium; Pyracantha yunanensis, P. gibbsi; Annona cherimola; Carissia grandiflora; Ceratonia siliqua, Diospyros kaki, Eucalyptus* sp., *Feijoa sellowiana, Fraxinus ornus, Persea americana, Olea europaea, Psidium guajava, Salix* sp., *Ulmus campestris, Quercus lobata.*

Nine inoculations on *Juglans nigra*, six on Sapote, and four on *Pterocarya fraxinifolia* were negative.

Biochemical changes accompanying curly top of tomato.—F. B. WANN and H. L. BLOOD.

A comparative study of the biochemical characteristics of healthy and curly-top tomato varieties showed that the diseased plants were considerably higher than the healthy in dry matter and dry weight of pulp, sugars, and solids in the juice and in freezing-point depression of the juice. The pH of the juice was not changed appreciably by the disease, but the titratable acidity of the leaf juice decreased and of the stem juice increased. Catalase activity was lower in the diseased leaves. Total sugar increased and total nitrogen decreased in the diseased leaves, resulting in a higher C/N ratio. In the diseased stems the total sugars and starch increased, which the total nitrogen decreased in some varieties but increased in others.

A comparison of the biochemical characteristics of healthy varieties with the degree of resistance to curly top revealed that, in general, those varieties with higher values were less resistant than those exhibiting lower values for dry weight, solids, sugars, and freezing-point depression.

The effect of vitamins on the growth of fungi in pure culture.—W. G. SOLHEIM, S. S. SEARS, and R. C. ROLLINS.

Preliminary studies on the effect of vitamins B and G on the growth of *Helminthosporium sativum, Aspergillus niger, Penicillium* sp., and *Phyllosticta* sp. have shown that fructification is markedly increased when either vitamin is added to corn-meal agar. Similar effects were observed when potato-dextrose agar was used but they were not nearly so pronounced. *Helminthosporium sativum* grown on corn-meal agar, alone, produced a very loose growth, while with the addition of either vitamin a very dense growth

was produced. In the potato dextrose the same type of growth obtained throughout and was similar to that on the corn-meal agar plus vitamin. In the checks the conidia were borne in groups on a conidiophore, while they were borne singly on the agar + vitamin G. *Aspergillus niger* and *Penicillium* sp. produced a deep orange yellow pigmentation with vitamin. No pigmentation was observed in the checks.

Studies of psyllid yellows of tomato.—H. L. BLOOD, B. L. RICHARDS, and F. B. WANN.

Psyllid yellows of tomato is of immediate importance only in so far as it becomes a confusing element in curly-top investigations. The symptomatology of the two diseases is similar, thereby necessitating differentiation. While heavy losses from this disease sometimes occur in localized areas, rarely do such losses constitute a problem of major importance. The disease produces a puckering of the younger leaves followed by a dwarfing, a slight curling upward and purpling of the margins, a thickening and grayish discoloration of the laminae, and a purpling of the veins. Twisting of the petioles and rolling of the older leaves are sometimes pronounced. Curly top induces more intense yellowing and leaf curling and more rapid death than psyllid yellows. Inoculations with fewer than 30 insects have consistently failed to induce the disease. As all plants that have been fumigated for the removal of the insects following the development of the disease have recovered, it is apparent that continued insect feeding is essential to the continued expression of the disease. These results give additional support to the insect-toxin theory of the etiology of psyllid yellows.

New symptoms of psorosis, indicating a virus disease of citrus.—H. S. FAWCETT.

In May, 1933, on citrus trees affected with psorosis, a clearing of small areas in young leaves giving a mosaic-like appearance was noticed for the first time. These clear regions seem to disappear as soon as the leaf approaches maturity, but, in some cases, rounded, clear spots are formed on some of the older leaves. These spots on older leaves vary much in size from mere dots to areas 10 to 15 mm. in diameter and often are accompanied by a slightly raised brown surface, occasionally in the form of rings. The spots on older leaves have been observed by the author for a long time. Herbarium specimens dotted with small spots were collected November 6, 1923, from young orange trees 18 months after they had been budded from older trees showing psorosis. These same trees first showed the bark-scaling symptoms 8 years after budding. This condition is quite distinct from mottle leaf. The transmission of the disease with its previously known scaly symptoms on the bark and these new symptoms on the leaves has been demonstrated by budding and by rooted cuttings. It is now believed that the disease may occur either as a localized scaly-bark form on the bark, without any leaf symptom, or as a systemic form with both bark and leaf symptoms. It is also suggested that two other citrus diseases, leprosis and ring blotch, with symptoms on older leaves of the same general nature, should be investigated as to the possibility of their virus origin.